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DISEASES DUE TO PROTOZOAN PARASITES.

SERGENT (Edm.), DONATIEN (A.) & LHÉRITIER (A.). **Des étalons guéris cliniquement de dourine peuvent rester des porteurs sains de germes pathogènes.** [Stallions Clinically Recovered from Dourine may remain (apparently) Healthy Carriers of the Pathogenic Organisms.]—*Bull. Soc. Path. Exot.* 1920. July 7. Vol. 13. No. 7. pp. 515–518.

During the course of the war a striking recrudescence of dourine occurred in Algeria, and the above workers from the Pasteur Institute of Algiers were called upon to undertake researches upon the methods of diagnosis and treatment of this disease. Affected stallions were handed over to the Institute by the military authorities, and, first of all, experiments were made in order to ascertain whether clinical recoveries could be confirmed by laboratory methods.

Microscopic examination of the blood of affected or recovered animals invariably showed no trypanosomes and thus recourse had to be had to the inoculation of large quantities of blood into a very susceptible animal such as the dog. A medium-sized dog would thus receive intraperitoneally about 200 c.c. of blood.

Stallions were chosen which had shown dourine lesions and had then been treated with a suitable remedy (atoxyl and orpiment according to MONOD's method). After regaining apparently all the signs of complete health these animals were tested periodically over a period of three years by extracting from each about 400 c.c. of blood at a time and this was inoculated into two dogs. The blood of each dog was afterwards examined three times a week for at least a year. Data are given concerning four stallions and these are summarised as follows :—

No. 1. During the month following upon complete clinical recovery its blood still infected two dogs. Subsequently 3½ litres of blood were inoculated, during three years, into 19 dogs, but no infection took place.

No. 2. After a double treatment the animal seemed to have made a complete clinical recovery. Over a period of a year 8 dogs received

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1½ litres of blood but were not affected. Fourteen months after the apparent recovery 1 out of 2 dogs became infected with the blood. Subsequently over a period of 2 years, a total quantity of 2 litres failed to infect 10 dogs.

No. 3. After treatment this animal seemed to present a complete recovery for a period of 2 years and was placed on active service with a cavalry regiment. 2,200 c.c. of blood taken during these 2 years did not infect 11 dogs. But, when tested again after this period of recovery the blood was found to be once more capable of infecting a dog.

No. 4. After 3 years and 3 months of complete recovery, during which time 3,640 c.c. of blood failed to infect in all 19 dogs, the blood again become infective for 1 dog out of 2.

It would therefore appear from these observations that horses cured completely of dourine from a clinical point of view, kept at active work and showing no trypanosomes in their blood even when tested by inoculation of several litres, suddenly again gave evidence of the presence of the organisms in their peripheral blood after intervals of 14 months, 2 years, and 3 years and 3 months.

A stallion that has been affected with dourine may thus remain a source of danger even when it has seemingly made a perfect clinical recovery.

Consequently all stallions that have been affected with dourine ought to be excluded from service at the stud. If the animal is in low condition it ought to be slaughtered. If, as the result of a suitable treatment, it regains apparent good health it ought to be castrated and it then may be capable of doing active work.

SERGENT (Edm.), DONATIEN (A.) & LHÉRITIER (A.). **Du diagnostic expérimental de la Dourine.** [Experimental Diagnosis of Dourine.]—*Bull. Soc. Path. Exot.* 1920. July 7. Vol. 13. No. 7. pp. 518–520.

Microscopic examination of the blood of horses affected with actively progressing dourine never revealed any trypanosomes. Microscopic examination of serous fluid from the characteristic plaques, from two typical cases, together with inoculation into mice and guinea-pigs likewise failed to show the organisms. The best method of demonstrating the organism is to inoculate large quantities of blood intraperitoneally into the dog as mentioned in the preceding extract. It is estimated that Arab and Barb horses kept at the Algerian studs contain each about 20–25 litres of blood. If 400 c.c. is taken for inoculation, from 1/50th to 1/60th of the total blood capacity is used for the test. This method enabled the authors to detect persistent infection in horses that were apparently quite recovered (see above). On the other hand, even when used in such high doses blood inoculations in certain cases failed to enable them to isolate the trypanosome from horses that presented a characteristic and complete clinical picture of dourine.

One stallion presented plaques and other characteristic lesions which indicated a relapse occurring over a year after the completion of medicinal treatment. Its blood. however, showed no signs of being infective for dogs (3½ litres inoculated into 19 dogs in all). A second

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affected stallion failed to infect 2 dogs with 400 c.c. of blood at the 2nd month of the disease, i.e., during the acute stage when it presented plaques. However, a dog became infected with 160 c.c. at the 6th month.

Two more stallions showed all the characteristic lesions of severe dourine, to which they both succumbed with typical symptoms in four months. The blood of one of these horses proved infective for a rabbit after the disease had been in progress one month but it gave negative results when inoculated into dogs on the 2nd and 3rd months (with 800 c.c.). The blood of the 2nd horse was not infective for dogs on the 2nd and 4th months (700 c.c.).

It would therefore appear that *T. equiperdum* is sometimes so rare in the general circulation of horses showing all the typical characters of very severe dourine—notably typical plaques—that an inoculation of susceptible animals with a quantity of blood equivalent to 1/50th or 1/60th of the total blood volume remains without result. The only results that are of any value for diagnostic purposes are, therefore, the positive ones when they are obtained.

SERGENT (Edm. & Et.) & DONATIEN (A.). **Infection expérimentale des dromadaires par le *Trypanosoma berberum* du debab.** [Experimental Infection of Dromedaries with *T. berberum*, the Causal Organism of *Debab*.]—*Bull. Soc. Path. Exot.* 1920. July 7. Vol. 13. No. 7. pp. 521–525.

An experimental study of debab in about 20 dromedaries was undertaken by the authors for a period of two years. The blood was examined, temperature taken daily, and the animals were submitted to no work but allowed to run free in a large paddock and well fed on straw and other fodder. (This sedentary mode of existence for the nomad and Sahara animals has the disadvantage, in the moist climate of Algeria, of facilitating greatly the development of mange, see p. 308.) The animals were first kept under observation for two months and then after they had been ascertained to be healthy they were inoculated subcutaneously with blood from a naturally infected dromedary.

The period of incubation was from 2 to 5 days. The average temperature of a healthy dromedary is about 37°C. Subsequent to the inoculation it commences to rise after 2–5 days and reaches or exceeds 40°C. The first rise precedes the appearance of trypanosomes in the peripheral blood by a day or two. With great regularity and in all stages of the disease a febrile crisis between 38° and 40°C occurred when trypanosomes made their appearance.

From the outset the trypanosomes made their appearance in large numbers in the blood. During the first days they might be regarded as "very numerous," i.e., as numerous as the red corpuscles or "extremely numerous," i.e., more numerous than the red corpuscles. It was exceptional to find on a slide trypanosomes that could be designated as "extremely rare." At all stages of the infection when the trypanosomes did appear in the peripheral blood they were in large number. When a superimposed affection (such as mange or pulmonary congestion) attacks a dromedary in which trypanosomiasis is latent the trypanosomes as the result of the lowering of the condition

invade the peripheral blood. The frequency with which the organisms were present in this situation was as follows :—during the 1st month from 22–25 days out of 30, during the 2nd month 16–20 days out of 30, during the 3rd month 10–15 days out of 31, 4th month 6–15 days out of 30, after the 4th month 2–4 days per month on an average (minimum 0, maximum 8 days). After 20 months the untreated survivors were still infected.

With regard to symptoms there was no distinct clinical sign which could be regarded as characteristic of debab. The general effect of the infection was a progressive weakness accompanied by considerable emaciation. The only striking and constant symptom was that the females aborted or gave birth to dead young camels. These accidents, however, only occurred during the acute stage which covered the first few months of the infection.

In a large proportion of these experimental cases the disease became, after passing through an acute stage, a latent infection which could not be detected 20 days out of 30 by means of the microscope, but could be demonstrated by the inoculation of large quantities of blood into susceptible animals such as dogs. During this stage the dromedary profits by a degree of relative immunity; it gains in condition and the females give birth to healthy young. Re-inoculation with the trypanosome only provokes a very slight febrile reaction and a very transient appearance of trypanosomes in the peripheral blood, and sometimes even no reaction whatever is produced. The transition into the chronic form is accelerated by the administration of certain curative agents. Two control dromedaries which were not treated had not fully attained this stage of latent infection with relative immunity after two years.

In this series of dromedaries the mortality rate was extremely low, and only one fatality could be ascribed undeniably to the trypanosome.

It is said by the natives that when the disease is contracted by the bites of tabanids it is nearly always fatal. However, in the conditions of experimental investigation referred to above trypanosomiasis was not sufficient in itself in the majority of cases to kill the dromedaries in spite of the extraordinary number of trypanosomes sometimes found by direct examination. The chief source of damage does not originate, it is stated, from the direct action of the trypanosomiasis but on account of its tendency to produce abortion and lowering of the general resistance of the body. Also, the proximity of infected dromedaries is a source of great danger for horses, mules, and donkeys; for, whilst in the case of the dromedary the disease may last many months and even end in recovery, in the equidae it only lasts a few weeks and always ends fatally.

SERGENT (Edm. & Et.) & DONATIEN (A.). **Deuxième note sur l'hérédité de l'infection et de l'immunité dans la trypanosomiase des dromadaires.**—[Second Note on the Heredity of Infection and Immunity in Trypanosomiasis of Dromedaries].—*Bull. Soc. Path. Exot.* 1920. July 7. Vol. 13. No. 7. pp. 525–527.

Two female dromedaries were inoculated experimentally with *T. berberum* on the 10th and 11th month of pregnancy. They gave

birth at full term (12 months) to dead young, the organs of which contained trypanosomes.

One of these two dromedaries and another one belonging to the same lot and inoculated on the same day were treated successfully for trypanosomiasis. Attacks of fever and presence of parasites became more and more rare and then disappeared. They were served about four or five months afterwards and in due time gave birth to healthy young. At this moment the camels could be regarded as clinically recovered but they still showed a latent infection with trypanosomes.

The blood of the young camels, however, was examined daily from the time of birth but showed no trypanosomes, and large quantities of blood were taken from them for test inoculations into dogs. The result was negative, indicating that the young born from recovered but still infected mothers were not themselves infected with trypanosomes.

It was then resolved to see whether these two young had any degree of hereditary immunity. They were both inoculated with infected blood, one succumbed to the infection in three weeks and the other in seven days. Young dromedaries therefore born from recovered but still infected mothers had no immunity against debab.

The authors conclude that during the acute phase of the disease the female dromedaries transmit the trypanosome to the foetus, which dies as the result of the infection. In the chronic phase of the disease characterised by a slight infection and a relative immunity the female dromedaries do not transmit to their progeny either their infection or their immunity.

LEGER (M.) & TEJERA (E.). **Contribution a l'étude du** *Trypanosoma venezuelense* **Mesnil 1910.**—*Bull. Soc. Path. Exot.* 1920. July 7. Vol. 13. No. 7. pp. 576–588. With 2 tables.

In 1905 RANGEL (Raph.) showed that the outbreaks of disease known under the names of *Peste boba* and *Desrengadera*, which cause high mortality in Venezuela among equidae, were caused by a trypanosome, which he identified with *T. equinum*, the causal agent of *mal de caderas*.

MESNIL in 1910 had sent to him blood smears from a horse by RINCONES and he showed that the parasite was morphologically distinctly different from *T. equinum*. Although he recognised that it recalled the surra trypanosome, *T. evansi*, MESNIL ascribed it to a new species *T. venezuelense*. DARLING at the same time described *T. hippicum* as the cause of *murrina* in the Panama Canal Zone. Recently Tejera undertook a further clinical study of the trypanosomiasis of Venezuelan equidae, and succeeded in infecting a certain number of other animals [see preceding number of this *Bulletin*, p. 175].

In the present paper the authors describe some further points with regard to the morphology of the trypanosome, its pathogenicity for experimental animals, and its drug resistance. From their observations and experiments they are led to believe that the disease

affecting Venezuelan horses is not surra. YORKE in fact had expressed the view that *T. venezuelense* was identical with *T. evansi*. The authors' results lent confirmation to MESNIL'S view, and it would therefore appear that *T. venezuelense* rightly constitutes a species in itself. Morphologically it resembles *T. evansi*, but it ought not to be confused with this trypanosome.

The authors' conclusions are somewhat as follows :—

T. venezuelense and *T. evansi* resemble each other morphologically but a careful comparative study shows that they are not identical.

The two trypanosomes do not set up in laboratory animals experimental diseases which present the same picture.

The therapeutic action of various arsenical products and tartar emetic is not the same in the two kinds of infections.

The two trypanosomes can be differentiated by means of cross-immunity tests applied after the administration of drugs. It appears, however, that the immunity set up by the surra organism is greater than that set up by *T. venezuelense*. This might be explained by the fact that the arsenical product employed, viz., the N. phenylglycylamide of arsenious acid, has evidently a stronger therapeutic action upon *T. evansi* than it has upon *T. venezuelense*.

The serum of a guinea-pig infected with *T. venezuelense* withdrawn during the period of crisis was found to have a rapid and pronounced trypanolytic action upon *T. venezuelense*, but none whatever upon the parasites of surra or of nagana.

DE KOCK (Gilles van de Waal). **Drug Treatment in Nuttalliosis.**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 637–675. With 32 tables.

No satisfactory method appears hitherto to have been devised for the treatment of piroplasmosis, or biliary fever, in equines due to the piroplasm *Nuttallia equi*. Treatment has been more or less empirical. It would seem that trypan blue is the drug which has been most frequently employed, and the results claimed to have been obtained with it have varied greatly. In South Africa biliary fever appears to be caused most frequently by *Nuttallia equi*, and in fact piroplasmosis due to the other type of parasite *Piroplasma* (or *Babesia*) *caballi* has only recently been demonstrated microscopically in the blood of a few horses, which were suffering from what appeared to be a mild form of the first-named disease. Smears showed distinctly rare paired pear-shaped piroplasms in the blood, similar to those commonly observed in bovine redwater and they were only present in the blood for a few days. Clinically these animals showed only very slight febrile symptoms.

In the researches described in this paper drug treatment was employed only on the type caused by Nuttallia parasites. The following were the drugs employed :—

(A) Inorganic salts.—(1) Sodium chloride ; (2) ammonium chloride ; (3) calcium chloride.

(B) Organic arsenicals.—(1) Arrhenal ; (2) arsenophenylglycine ; (3) salvarsan.

(C) Dye stuffs.—(1) Fuchsin; (2) gentian violet; (3) novoflavin; (4) trypan red; (5) methylene blue; (6) trypan blue; (7) brilliant green; (8) malachite green; (9) crystal violet.

(D) Other drugs and preparations.—(1) Iodine; (2) formalin; (3) nucleinic acid; (4) quinine hydrobromide; (5) electrargol; (6) camphor; (7) turpentine; (8) thymol; (9) creosote.

In many tests several of these drugs were used upon the same animal and were injected either simultaneously or at varying intervals of time. The horses employed were, (1) naturally affected horses, i.e., horses which had not been inoculated experimentally previously; (2) horses that had become affected with biliary fever during or after immunisation against horse sickness; (3) horses which had become affected after hyper-immunisation against horse sickness, a process in which the animals received by transfusion large quantities (about 10 litres) of virulent horse sickness blood.

Cases were usually detected when they occurred on account of a rise in the daily temperature, which was taken as a routine procedure twice daily in the Laboratory. Affected animals could therefore hardly escape observation for more than 24 hours. In all cases where biliary fever was suspected a microscopical examination was made almost immediately so that they were treated as soon as possible. No cases were treated unless *N. equi* was first demonstrated in the blood. This is regarded as of fundamental importance inasmuch as biliary fever often resembles some forms of equine infectious anaemia. The severity of the cases met with varied greatly. In some the onset of the disease was so sudden that haemoglobinuria was the first symptom to attract attention. This often happened while an animal was passing through a horse-sickness reaction during the process of immunisation. Severe cases were also brought in from farms for treatment, and some of these animals had probably been ill for a few days before the symptoms had become alarming. In other cases the disease was less rapid in its onset and became more acute on subsequent days. Some cases were so mild that very few manifest symptoms were seen.

After injection of the drug the animals were usually kept under close observation and blood smears examined at intervals in order to ascertain the progress of the disease. The injection was repeated as soon as it was found that the first one had no appreciable effect, or the first drug was substituted by other drugs, and in some cases more than two drugs were used. The animals were always kept in well-ventilated places and specially dieted. Symptoms such as anaemia and collapse were treated with intravenous injections of saline solutions, defibrinated blood, or injections of caffein, strychnine, camphor oil, strophantin, and other stimulants, The usual doses of the various drugs were employed but the safe and maximum dose that could be employed with impunity was often first of all determined on normal horses. It frequently resulted that much larger quantities of the drug could be given than had been prescribed by other investigators; for example, with quinine acid-hydrobromide about 10 times the dose employed by WILLIAMS (A. J.), 1914, in India, could be given. The following composite table summarises the results obtained.

DE KOCK. COMPOSITE TABLE.

		Drug.	Animals treated.	Parasites frequent.	Parasites rare.	Recoveries.	Deaths from Nuttalliosis.	Deaths from
A. Inorganic Salts.	1	*Sodium chloride*	1	1	—	—	1	
	2	*Ammon. chloride*						
		+ Calc. chloride	2	2	—	—	2	
		+ Gentian violet	1	1	—	—	1	
	3	*Calcium chloride*	8	6	2	6	2	
		+ Ammon. chloride : see above						
		+ Meth. blue	2	2	—	1	—	1
		+ Quinine hydro-C. + Trypan blue	1	1	—	—	1	
B. Arsenicals.	1	*Arrhenal*	2	1	1	1	1	
		+ Meth. blue	3	2	1	1	—	2
		+ Quin. Hydrob. + Trypan blue	2	2	—	1	1	
	2	*Arsenophenylglycine*	9	5	4	5	4	
		+ Trypan blue	20	17	3	10	10	
		+ Meth. blue	2	1	1	1	—	1
		+ Meth. blue and Quin. Hydrob.	1	1	—	1	—	
		+ Trypan blue and Quin. Hydrob.	1	1	—	—	—	1
		+ Trypan blue and Nucleinic Acid	1	1	—	—	1	
		+ Trypan blue + Brill. Green + Malach. Green	1	1	—	—	1	
	3	*Salvarsan*	3	2	—	2	—	1
C. Dye Stuffs.	1	*Fuchsin*	1	1	—	—	1	
	2	*Gentian violet*						
		+ Ammon. Chlor. See Ammon. Chlor. above.						
	3	*Novoflavin*	1	1	—	—	1	
	4	*Trypan red*	2	2	—	—	1	1
	5	*Methylene Blue*	2	1	1	—	1	1
		+ Calc. chloride	2	2	—	2	—	
		+ Arrhenal	3	2	1	2	—	1
		+ Arsenophenylglycine	2	1	1	1	—	
		+ Arsenophenyl + Quin. Hydrob.	1	1	—	1	—	

MPOSITE TABLE.

REMARKS.

d following day.

ed in 5 days, and 1 in 5 days from sequelae.
d in 6 days. Ammon. chloride in conjunction with these drugs either by drenching or subcut. (8 gm.) seems to have had no effect in these 3 cases.

irst Calcium Chloride seemed to give good results, but subsequently this drug even njection with others did not have the desired effect. The doses ranged from 10–20 grams ed in fairly large quantities of water, and injected intrajugularly. In the majority cute symptoms of shock were observed directly after injection or infusion. In one t was fatal. The chief symptoms of shock were more or less as follows; the animal omes to shiver, which quickly spreads over the whole body, followed by uneasiness, g from side to side, snorting, blowing the nose and an anxious facial expression. These oms usually last several minutes, followed by some depression afterwards. It might nted out here that in the majority of cases treated with Calcium Chloride, and also case of a few other drugs, the parasites after treatment assume contracted forms, which ally may become a rounded structureless mass of chromatin (like Anaplasma). In one econdary Anaemia followed, which was most probably due to the injection of the thylene Blue, but this will be more fully discussed later on.

es utilised were 5–10 grams dissolved in 500–1,000 c.c. of physiological water, and in- intrajugularly. In some cases slight symptoms of shock were also observed. In two here was secondary anaemia as a result of the Methylene Blue.

es ranged from 5 to 22 grams dissolved in a couple of litres of normal saline and d intrajugularly. Sometimes the injection was repeated. In some cases it was used ther drugs as an haematinic. Some of the cases were complicated with Horse-sickness, sult of immunisation, followed by a high fever, but in which symptoms of Nuttalliosis ed to be fairly mild. Again cases occurred with Secondary Anaemia as a result of the n of Methylene Blue.

and 10 gm. respectively were injected subcut. Both the cases which were injected ed to be fairly mild. This treatment needs to be repeated as soon as more salvarsan e obtained. The horse which died of shock received 10 gm. intraven.

d in 3 days. 1 gramme intraven. on 1st and 2nd days.

d in 3 days. 3½ grammes intravenously.

c.c. of 3 per cent. and 2 per cent. Trypan red injected subcutaneously respectively.

thylene Blue in large doses (5 grammes) intrajugularly seems to have good parasiticidal on *Nuttallia equi*, but it produces secondary anaemia, resulting in death in many cases. is drug has this latter effect was shown in controls, and in subsequent experiments, ble of which will be discussed in another paper [see p. 351]. Smaller doses were tried t success.

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COMPOSITE

	Drug.	Animals treated.	Parasites frequent.	Parasites rare.	Recoveries.	Deaths from Nuttalliosis.	Deaths from other causes.
C. Dye Stuffs—*contd.*	6 *Trypan Blue*	31	27	4	12	19	—
	+ Arsenoph. Glycine (+ 1 with Nuclein. Acid)	2	17	4	9	11	1 shock
	+ Arsenoph. Glycine + Quin. Hydrob.	1	1	—	—	—	1 do.
	+ Calc. Chloride + Quin. Hydrob.	1	1	—	—	1	—
	+ Arrhenal + Quin. Hydrob.	2	2	—	1	1	—
	+ Brill. Green + Malach. Green	4	4	—	—	3	1 shock
	7 *Brilliant Green etc.* + Malachite green, etc. + Crystal violet, etc.	see also in conjunction with Trypan Blue and Arrhenal.					
D. Other Drugs and Preparations.	1 *Iodine* + Formalin	1	1	—	—	1	—
	2 *Formalin* + Iodine (see Iodine)						
	3 *Nucleinic Acid* + Trypan Blue + Arsenophenylglycine	see Arsenophenylglycine					
	4 *Quinine Hydrobromide*	12	2	10	9	2	1 sh
	+ Arsenophenyl + Trypan Blue	1	1	—	—	—	1 do.
	+ Arrhenal + Trypan Blue	2	2	—	1	1	—
	+ Calc. Chloride + Trypan Blue	1	1	—	—	1	—
	+ Arsenophenyl + Meth. Blue	1	1	—	—	—	1 To anae
	5 *Electrargol*	2	2	—	1	1	—
	6 *Camphor*	1	1	—	1	—	—
	7 *Turpentine*	1	1	—	—	1	—
	+ Camphor	1	1	—	1	—	—
	8 + *Thymol* + Turpentine						
	9 + Creosote						

ABLE—*contd.*

Deaths from other causes.	REMARKS.
—	ctions of 1 per cent. and 2 per cent. were utilised, and in some cases subcutaneously but majority intrajugularly. Doses ranged from 100 c.c. to 400 c.c. and in one case the n was repeated as many as four times. There were several cases complicated with ckness as the result of immunisation. The cases of shock which proved to be fatal e to the other drugs used in conjunction with the Trypan Blue.
1 shock	
1 do.	
—	
1 shock	
Trypan Blue	se three drugs, Brilliant Green, Malachite Green, and Crystal Violet, might be con- together. Control horses were first treated to ascertain the safe dose intrajugularly. found that 100 c.c. of a 1 per cent. solution, either of Malachite Green or Crystal could be utilised, but in the case of Brilliant Green this dose produced moderately acute ms of an intoxication, such as slight colic, sweating, salivation, etc. The dose for the was accordingly reduced to 100 c.c. of ½ per cent. solution. These three drugs when and injected into one case of Billary Fever caused acute symptoms of shock and When this was subsequently controlled in a normal horse, acute symptoms of an ation followed the injection.
—	d in 2 days. 1st day 3 grams. iod. intraven. 2nd day 5 c.c. formalin intraven.
1 shock	
1 do.	
—	
—	
1 Trypan Blue and Anæmia	ther Quinine Hydrobromide or Quinine Bi-Hydrobromide was used intrajugularly in ranging from 5–10 grams dissolved in 500 c.c.–1,000 c.c. normal saline. In some the was repeated as many as three times. On account of the alarming symptoms of shock ing in the death of a few animals, the dose was fixed at 5 grams. The symptoms of shock scribed by Williams (see above) correspond, but such symptoms are not only confined es with high temperatures, but also occurred in normal horses. om the table it would appear that fairly good results were obtained with this Quinine but it must be remembered that 10 of the 12 cases treated were fairly mild, and that o acute cases died in spite of treatment. One animal died as the result of Pernicious mia and Secondary Fever due to the Methylene Blue.
—	tal case 5 c.c. at once and 5 c.c. 5 minutes later on 1st day, 5 c.c. again on 2nd, 3rd, and ay. Died on 5th day. Recovered case 5 c.c. 1st day and on 2nd day ; fever fairly acute rmal again on 6th day.
—	.c. camphor sol. + 20 c.c. alcohol in 1 litre water intraven. 1st day, ditto 2nd day. ugh parasites frequent symptoms not acute.
—	t day 10 c.c. camphor sol. + 80 c.c. alcohol in 1 litre water intraven. 2nd day 5 c.c. ne + 40 c.c. alcohol in 1 litre of water. 3rd day 5 c.c. thymol + 40 c.c. alcohol in 1 litre ter intraven. 4th day 5 c.c. turpentine + 40 c.c. alcohol in 1 litre water intraven. r acute case with haemoglobinuria. On the 5th day there was steady decrease in temp. alse. On 7th day temp. normal but still symptoms of anaemia. Camphor, turpentine, l and creosote were also used. These substances were dissolved in hot alcoholic n, and infused at blood heat. A number of control horses were first subjected to the gular injection of 1 litre alcoholic solution of different strengths to ascertain what con- tion of alcohol could be injected with impunity. It was found that one litre of a r cent. sol. was quite safe, whereas 1 litre of a 50 per cent. sol. caused shock, but with ry, and 1 litre of a 75 per cent. solution, shock and death. In the case of camphor, turpen- thermol, and creosote a dose of 5 c.c. was first tried on a number of control horses, ose was gradually increased until the drug tested produced symptoms of an intoxication. this a safe dose was determined.

Before assessing the relative value of the different drugs utilised de Kock mentions an all important factor relevant to this point, viz., that many mild cases of biliary fever, naturally occurring or otherwise, do recover completely without the interference of any medicinal treatment. Such recoveries even took place where parasites were present in somewhat considerable numbers in the blood smears; therefore it could hardly be considered a fair test of the drug when treatment was undertaken in cases showing slight symptoms and where the number of parasites remained rare throughout the whole course of the disease. Much of the value of any particular drug tested would depend upon the severity of the case, the number of parasites present in the blood smears, the progress of the disease, and the disappearance of the parasites after treatment.

The author maintains from his experience that all the drugs employed, in more than 100 cases, tabulated above, gave unsatisfactory results, with one exception, viz., methylene blue. Even this drug had eventually to be discontinued on account of a secondary anaemia which it produced, often with fatal results. [See p. 351.]

Further he concludes that the divergent opinions of different investigators as to the success of certain drugs might be explained in one or other of the following ways :—(a) Cases of biliary fever in South Africa might easily have been mistaken for certain forms of infectious anaemia, and in such cases the drugs might appear to have produced distinctly beneficial effects. For, it is known that many cases of infectious anaemia during the periods of remission show symptoms like those of biliary fever and without any treatment either recover completely or subsequently show further remissions and symptoms which usually become less severe and less noticeable. (b) There may be different strains of Nuttallia, some of which are less virulent than others and therefore less resistant to some of these drugs. [But it has been proved that the drug resistance and virulence of certain protozoan parasites are not directly related (see, for example, this *Bulletin*, 1918, Vol. 6, No. 2, p. 67).—Ed.] (c) In South Africa very likely *P. caballi* infection has been treated instead of the more virulent Nuttallia type.

In the meantime it is suggested that quinine hydrobromide should be used for mild cases of Nuttallia infection as soon as they are detected and that further experiments with other drugs ought to be carried out on the more severe cases.

UNION OF SOUTH AFRICA. **Report from the Select Committee on the Spread of East Coast Fever in the Union, together with the Proceedings of the Committee, Minutes of Evidence, and Appendix.** 1920. July 15. xvi–152 pp. f'cap.

A Select Committee was appointed by the Senate of the Union Parliament on the 26th May 1920 " to enquire into and report upon the unabated spread of East Coast fever in the Union and especially to consider what remedies, and their application, will best cope effectually with the great loss to the farmers of the Union caused by the continued sporadic outbreaks of this disease."

Evidence was obtained from a large number of farmers, professional men, and officials who had been in close contact with East Coast fever for a number of years.

It appears that some 18 years ago East Coast fever was introduced from Portuguese East Africa, through Rhodesia, into what is now the Union. Crossing the Limpopo, it first broke out in the District of Zoutpansberg. At that time nothing was known with regard to the nature of the disease and its devastating effects quickly spread southwards.

The prevalence of the disease in the Union and the adjoining Native Territories has cost the country, and the cattle farmers especially, millions of pounds. The Committee are now of the opinion that it will be far better to incur increased expenditure for a shorter period than to allow the scourge to continue any longer merely for the temporary advantage of economy. Given the necessary expenditure of public funds and the intelligent and adequate administration of the existing regulations it is considered possible to stamp out the disease in two years.

With regard to the remedial measures adopted in the past the Transvaal Crown Colony Government originally attempted to stop the disease by fencing off large areas of land, but as there was no organised check on the movement of stock the disease spread unhindered. When it was realised that fencing was not effective the Government resorted to the expedient of killing off herds, large and small, whenever there was an outbreak. The result of this was that the northern districts of the Transvaal became almost denuded of cattle. However, when the outlook was very black an enterprising farmer, the Honourable Joseph BAYNES, of Natal, proved the efficacy of dipping and hand-dressing of cattle to destroy ticks. Later, WATKINS-PITCHFORD further investigated this question and proved that the only practicable remedy for the disease lay in the systematic dipping of cattle at short intervals in a solution that would kill the tick. This view was then corroborated by THEILER'S work at the Government Laboratory, and since that time the disease has been checked in a measure by the action of the more intelligent farmers who have erected dipping tanks, and thereby saved their herds.

Recently a great measure of success has been obtained in eradicating the disease from Swaziland and the Transkeian Territories. In Swaziland the Principal Veterinary Officer [ELDER W. A.] has, through the co-operation of farmers and others, succeeded in stamping out the disease in the short space of two years.

The evidence showed that the administration and the carrying out of East Coast fever regulations leave much to be desired, and it is difficult to determine whether this is caused by want of interest in the subject, by fear of offending those farmers who have no faith in dipping, or by want of funds to carry out the regulations. However, the fact remains that in many areas the disease has been allowed to spread unchecked. This serious state of affairs shows culpable inefficiency in the Public Service and also largely want of co-operation between officials and the farming community.

Attention is drawn to the necessity of instituting an active educational propaganda. The evidence, it is stated, furnished overwhelming proof of the efficacy of dipping and hand dressing as recommended by WATKINS-PITCHFORD. The eradication of the disease by this method requires, however, thorough administration in the application of the regulations. The present method of forwarding blood smears for

examination could be much improved upon, for a speedy diagnosis is invaluable. The present loose manner which seems to prevail in the issue of permits, with consequent inability to trace movements of cattle, accounts for the majority of the sporadic outbreaks. While admitting the usefulness of fencing of farms the Committee is of opinion that taking into consideration the immense area infected, in the Transvaal especially, it would be ruinous to farmers to enforce compulsory fencing, and such a course should be resorted to in extreme cases only.

In order to prevent the further ravages of the disease the Committee makes the following recommendations to the Government :—

" (a). Establishing a separate section in the Agricultural Department for the special and express purpose of combating with and preventing the outbreak of East Coast Fever, and appointing a capable, energetic and firm official with wide experience of the disease as the head of such Section, who shall be responsible directly to the Minister for the carrying out of the Regulations.

" (b). Placing the local administration of the tick fever regulations in the hands of the Resident Magistrate of the District.

" (c). Making the local Veterinary Officer advisory to the Magistrate (thereby relieving him as far as possible from administrative office work) and providing him with motor transport to get into closer touch with farmers, and not only to perform the proper functions of his office in tendering advice, but also to check the field administration of the Magistrate.

" (d). Selecting as inspectors and cattle guards men of intelligence who shall be trained in the duties pertaining to the eradication of East Coast Fever and particularly to the methods for securing the efficiency of dipping tanks, with special instructions to scrutinize carefully all cattle within the areas and ensure that they are properly dipped. Such officials should be sworn in as policemen and be subject to the Police Regulations. Incentives, in the way of promotion, should also be held out to them as a reward for vigilance and zeal in the execution of their duties.

" (e). Making compulsory the building of dipping tanks in areas liable to East Coast Fever (not only on private farms, but also on Crown lands and native locations), and in that connection giving the greatest encouragement possible in the erection of such tanks, by means of advances through the Land Bank under the existing law, and also by assisting farmers to secure materials for building the tanks at the cheapest rates.

" (f). Having all dipping tanks inspected and tested regularly at least once a month, and giving greater facilities for the testing of the liquid in such tanks.

" (g). Taking immediate steps in order that isometers, which are now almost improcurable, be placed within the reach of every cattle farmer at cost price.

" (h). Providing every District Veterinary Officer with a microscope in order that he may quickly investigate and report upon the bloodsmears sent to him.

" (i). Instituting a regular system of analysis of dipping materials offered for sale, the vendor to be under special licence and his wares to be subject to analysis by the Department at all times for strength and solubility, and that in regard thereto the Adulteration Act be strictly enforced.

" (j). Setting on foot an extensive East Coast Fever propaganda.

" (k). Instituting a complete and effective check upon the movement of cattle in and around infected and suspected areas by insisting :—

" (i). That permits shall only be issued by the Magistrate of the District, who may, however, for the convenience of the public, depute trustworthy and responsible officials entrusted with the carrying out of the East Coast Fever Regulations to issue movement permits in his name, and that such officials be made readily accessible by the public immediately when required, their names to be published in a local paper as well as posted up at prominent centres in the district.

"(ii). That it shall be wholly illegal to grant a permit for the movement of cattle out of or through an infected or suspected area, and that all cattle found under such illegal movement shall be instantly destroyed and the offender prosecuted.

"(iii). That a proper register of cattle movements under permit shall be kept and a copy thereof sent monthly to the head of the East Coast Fever Department, who shall closely scrutinize it.

"(l). Importing fencing material and placing it at the disposal of farmers at cost price.

"(m). Discontinuing the practice of killing off cattle as being ruinous and unnecessary."

LAVERAN (A.) & FRANCHINI (G.). **Infections expèrimentales de chiens et de cobayes à l'aide de cultures** d'*Herpetomonas* **d'Insectes.** [Experimental Infection of Dogs and Guinea-pigs by means of Cultures of Insect Herpetomonads.]—*Bull. Soc. Path. Exot.* 1920. July 7. Vol. 13. No. 7. pp. 569–576. With 1 fig.

In previous papers the authors demonstrated that it is possible to produce serious and even fatal infections in white mice by inoculating them with flagellate protozoa from various insects [see this *Bulletin*, 1920. Vol. 8. No. 1. p. 17.].

In this paper a summary is given of a number of experiments of the same kind performed upon dogs and guinea-pigs.

Dogs. Experiments were undertaken on two dogs which were inoculated with cultures of *Herpetomonas phlebotomi* (i.e., the flagellate from the sand fly).

In one of these dogs, that had been inoculated in three different places under the skin of the outside of the right thigh, boils appeared at the points of inoculation. These boils bore a great resemblance to oriental boil and they showed no tendency to suppurate. Material was taken from these boils or buttons by puncturing several times, and free-lying and intracellular parasites were seen which bore the greatest resemblance to *Leishmania tropica.*

The boils developed eight days after the inoculation and lasted 54 days. After recovery the dog was re-inoculated with cultures of the same species of flagellate but the result of this inoculation remained negative. It would therefore appear that the dog had acquired an immunity against the organism.

On no occasion were parasites found in the circulating blood of the dog and the infection thus remained localised. The second dog inoculated was a very young one, and the injection was made on the outside of the thighs using a virus of the same kind as was used for infecting the above dog. In this case a generalised infection was produced without local lesions, a result attributable undoubtedly to the fact that this dog was more sensitive to the virus on account of its being much younger than the first dog. No change whatever was produced at the points of inoculation and material obtained by puncture of these points only once disclosed a Leishmania-like parasite. On the other hand, parasites were seen in the peripheral blood on several occasions, and three months after the original inoculation the dog showed symptoms of illness, viz., wasting, anaemia, general weakness, and unsteady gait. The dog was killed in very poor condition and on post mortem examination it was found that the liver, spleen, and bone marrow contained lesions very similar to those of kala-azar. There

were present, however, elongated elements resembling Herpetomonas in appearance, but devoid of flagella; these were nevertheless rare in comparison with the Leishmania-like parasites.

Guinea-pigs. Three guinea-pigs were inoculated with cultures of *H. ctenocephali* (the flagellate of the dog flea), three more with a variety of this parasite described by CHATTON [see this *Bulletin* 1919, Vol. 7, No. 3, p. 151], and three more with cultures of *H. phlebotomi.* Out of these nine guinea-pigs eight were inoculated intraperitoneally and the remaining one was inoculated into one of the testicles.

Of the eight inoculated intraperitoneally one only died, viz., a very young guinea-pig weighing 80 grammes, and therefore less resistant to the infection than an adult guinea-pig. This young animal was inoculated twice at three days' interval with large doses of *H. ctenocephali.* Leishmania-like parasites were observed in its blood during life, and, after death, in the peritoneal exudate, liver, spleen, and bone marrow.

The seven other guinea-pigs inoculated intraperitoneally showed a mild form of infection and they were killed in apparent good health in from 44 to 113 days after inoculation, and it was found that they were still almost all infected. The three viruses employed had produced identical results. The infection was not manifested as a rule by any symptom. It was only revealed by histological examination of the blood. Leishmania-like parasites appeared in the blood in from 2 to 6 days after intraperitoneal inoculation. They were very rare at first but increased in number after a few days. They subsequently disappeared from the peripheral circulation but their presence was still found for a fairly long time in the viscera. On post-mortem examination a slight increase in size of the spleen was often found, and the peritoneum, which was sometimes congested, contained a small quantity of turbid serous fluid, which contained Leishmania-like parasites in varying numbers. The parasites were also visible in most cases in stained smears made from the liver, spleen, and bone marrow. Usually it was in the spleen that they were least rare. Alongside the Leishmania-like parasites which preponderated and were sometimes free-lying and sometimes intra-corpuscular there were often found elongated parasites with nuclei and centrosomes, recalling Herpetomonas in shape, but without flagella.

A mouse inoculated with peritoneal exudate from one of the guinea-pigs became infected and rapidly succumbed.

The guinea-pig which was inoculated into the testicle received a quantity of culture made from CHATTON'S variety of the dog flea flagellate. A localised infection was produced limited to the inoculated testicle but no indication of generalised infection was noticed to take place; examination of peripheral blood always gave negative results. The guinea-pig was killed 117 days after inoculation and its inoculated testicle was then completely atrophied. It is remarked that Leishmania inoculated in the form of culture or otherwise into the testicles or into the peritoneum of mice gave rise frequently to lesions in the testicle leading to atrophy of these organs. Thus we have still further analogy between the lesions produced by these parasites and those which are seen to occur as the result of infection provoked by means of *Herpetomonas.*

MARULLAZ (M). **Sur l'évolution de** *Sarcocystis muris.* [The Life Cycle of *Sarcocystis muris.*]—*Ann Inst. Pasteur.* 1920. Aug. Vol. 34. No. 8. pp. 547–552. With 31 text-figs.

A considerable amount of information upon the development of *S. muris* in the intestine has been recently contributed by SMITH (Theobald) 1901, NÈGRE (L.) 1907, 1910 and 1918, and CRAWLEY (H.) 1914 and 1916. The manner in which the parasite develops after it has penetrated into the intestine of the infected animal still remains to be elucidated. NÈGRE claims that the parasite undergoes cyst formation and that the cysts are passed out of the body with the excreta and are then capable of carrying on the infection into healthy animals. CRAWLEY believed that he had demonstrated the existence of macrogametes, microgametes, and fertilisation within the cells of the intestinal villi [see this *Bulletin*, 1916. Vol. 4. No. 4. p. 148.] ERDMANN (1910, 1914) observed an amoeboid form of the parasite which penetrated into the lymphatic spaces of the intestine in the new host, then passed into the adipose tissue of the body and reached the muscles in the form of small binucleated elements; the number of nuclei multiplied rapidly until ultimately the so-called Miescher's tubes were formed.

In the adult stage *S. muris* has the appearance of whitish threads which may measure up to 2 cm. long and are disposed lengthways among the muscle fibres. The filaments are divided up into small elements from 1 to 2 mm. long by 0·25 mm. broad with rounded extremities. These structures, which the author designates as typical tubes, are made up of a structureless cuticle from which run fine partitions into the lumen of the tube. In the compartments enclosed by these partitions are found a large number of spores varying in shape and size. Some are rounded and 4–5 microns in diameter, others are elongated or crescent-shaped and measure on an average 10–11 microns by 5–6 microns. Large binucleated oval or regularly crescentic forms measuring up to 16 microns long by 9 microns broad are also found. In the case of a long-standing infection all the striped muscle tissues are invaded by the parasite. On the other hand the parasite was never found by the author in the heart muscle.

When infected muscle has been fed to clean mice one can find the spores in the epithelial cells of the small intestine in $1\frac{3}{4}$ hours and the spores take the form of oval-shaped elements lodged in the upper part of the cell. The cytoplasm stains very slightly rose-coloured when stained by haematoxylin-eosin and no metachromatic granules are seen; the nucleus is centrally placed and is distinctly granular in structure. The host cell at this stage does not seem to undergo any change apart from an increase in size due to the presence of the parasite. Usually after a short time, however, the nucleus of the epithelial cell loses its elongated form, becomes rounded, and is pushed towards the basal membrane whilst at the same time it undergoes pyknosis, but the cytoplasm presents no appreciable reaction when stained by the usual methods.

From the second hour onwards the spore commences to develop, it becomes rounded in shape and its cytoplasm, although it remains clear, seems to diminish in size, its nucleus loses its granular structure and the chromatin becomes arranged in the form of small rods with a

radial arrangement. This indicates commencing mitosis and it is further manifested by the formation of polar bundles which ultimately become separated completely towards about the 4th hour after the ingestion of the parasite. It is rare to see the cytoplasm undergo division subsequently to the nucleus in these intracellular bodies. Nevertheless one may often see within the lumen of the intestine sarcosporidia that are free or included within the desquamated epithelial cells and they are binucleated and similar in appearance to those observed in the covering of the villi. In the intestinal lumen one may also see sarcosporidia that are crescentic in form and have a single nucleus similar in appearance in every respect to the large forms found in the muscles. These forms are produced by the division of the above binucleated forms. These elements are not identical with those which were used for infecting the animal for it is towards the 10th day after the experimental feeding that they become most numerous.

Although the sarcosporidia that remained within the epithelial cells presented all the stages of mitosis others were seen which, after the commencement of this process, in the diaster stage, left the host cell and penetrated within the lymphatic spaces of the tissue underlying the villi. These sarcosporidia are of normal size each with a nucleus which is gourd-like in shape and composed of two rounded masses, homogeneous in structure and thus probably indicating a cessation of mitosis. These elements were observed to penetrate within the lymphatic spaces in from the 24th hour after infection and no change in their appearance was noticed to occur up to the point when they had reached within the muscular coat of the intestine.

The small size of the mesenteric lymphatics and glands in the mouse unfortunately made it impracticable to examine preparations from these tissues, but further development was traced by examination of the liver. In this organ the parasites were found from the 11th day onwards in the form of small oval elements measuring 4 by 3 microns with a homogeneous cytoplasm which stained clear blue with Giemsa and contained a rounded rod-shaped karyosome which was excentrally placed. These organisms were either free-lying or included within the hepatic cells. They were also found in the spleen but here they they were less numerous and always extra-cellular.

Examination of the muscle tissue disclosed the presence of structures analogous to those found in the liver between the 44th and 55th day after the original feeding. There were other forms with the same cytological peculiarities, but a little more elongated or crescentic in form. These parasites were either found within the muscle fibres or about to penetrate into them. Besides these forms with a single nucleus there were others which were more rounded in shape and possessed two karyosomes which were clearly separated from each other, indicating that a process of division had commenced. In addition a round intramuscular element was observed 5 microns in diameter and enclosing 8 similar bodies disposed in rosette-shaped fashion within an amorphous and unstained substance. Each of these small bodies was composed of a homogeneous protoplasmic mass containing a ball of chromatin.

In considering the above observations the author concludes that it is reasonable to admit that a large number of the free-lying sarcosporidia in the intestine are expelled with the faeces, and may thus serve to

propagate the infection among mice living together with the experimental animal. Cysts analogous to those described by NÈGRE were not observed but it is possible that the spores thus evacuated represent a resistant stage.

With regard to the remaining sarcosporidia which travel into the lymphatic spaces of the intestine, then through the viscera (liver and spleen), and then reach the muscle tissue where they multiply and form daughter spores it is admissible that they represent the initial stages in the formation of a Miescher's tube.

In all, 65 mice were examined between the first hour and the 127th day after experimental infection, but nothing was seen to suggest fertilisation of the spores included in the intestinal cells as pictured by CRAWLEY.

As has happened with other investigators numerous attempts to infect animals by intraperitoneal and intramuscular inoculations failed completely.

WALKER (James). **Some Observations in Connection with the Occurrence of "Sarcosporidia" in the Skeletal Muscles of Sheep and Horses in South Africa.**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Agriculture.* 1918. April. pp. 397–424.

Sarcosporidia have been described in almost all countries and in almost all the domesticated animals and certain authors have attributed to them a pathological significance (notably MCGOWAN as regards scrapie in sheep, and WATSON, certain cases of lameness in horses). Under THEILER'S direction Walker carried out the series of examinations described in this paper in order to determine (1) to what extent sarcosporidial infection occurs in the muscles of horses and sheep in South Africa, and (2) whether the parasite could be considered responsible for any symptoms or pathological conditions particularly of the muscular or nervous systems. The so-called sarcosporidial tumours (*Balbiana gigantea*) found on the oesophagus of the sheep were not taken into account in the course of the investigation.

Portions of various muscles, comprising the gluteus, quadriceps extensor cruris, supra-spinatus, triceps extensor cubiti, longissimus dorsi, psoas, masseter, and splenius, were collected from a number of sheep of various ages ranging from six weeks old to eight-toothed sheep. Portions of the sciatic nerve were also examined. In the same manner portions of like tissues were subjected to examination from a number of horses of various ages that had died or were killed at the Onderstepoort laboratory.

The specimens collected were fixed in 10 per cent. formalin and sections measuring ½ inch square were made from each of these by means of the freezing microtome, stained with haematoxylin-scharlach, and examined microscopically.

Altogether specimens were collected from 27 sheep and 26 horses that were under observation at the Laboratory in connection with various experiments. The number of cysts observed together with their measurements are detailed in a series of tables.

Cysts were found in all the sheep examined with the exception of one—a six weeks old lamb, and in all the horses with the exception of three.

With regard to the sheep the greatest number of cysts observed in a single section was 40, and 331 in sections from all the above-named skeletal muscles in a single sheep. Fatty degeneration was detectable in the muscle tissue but no changes in the peripheral nerves.

In sheep in poor condition an oedematous condition of the intermuscular connective tissue was sometimes found. The symptoms described by McGowan as characteristic of the disease known as scrapie, viz., pruritus, loss of wool, and loss of muscular power were not observed in these infested sheep. The poor condition was associated, in some of the sheep, with a verminous infection (due to *Haemonchus contortus* and *Oesophagostomum columbianum*).

In the horse the highest number of cysts found in a single section was seven, and in sections from all the above-mentioned muscles 45. When these animals had suffered from horse sickness and pernicious anaemia, piroplasmosis and toxaemia, a fatty degeneration could be seen in the muscles but in such cases the degeneration could naturally be attributed to the effect of the toxins produced by the causal agents of these diseases. In some of the horses, particularly the horse sickness affected ones, the intra-muscular tissue was oedematous. No changes were noted in the peripheral nerves of any of the horses with the exception of three that were affected with dourine; in these three cases a neuritis was discernible and moreover these particular cases showed little or no sarcosporideal infestation.

The symptoms which are associated with the disease known as lamziekte in cattle were not observed in any of these sheep and horses.

Viljoen (P. R.). **On Sarcosporidiosis in Relation to Lamziekte.**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. April. pp. 453–576. With 5 plates comprising 10 figs. & 100 tables.

The study of sarcosporidia in relation to lamziekte in cattle was taken up as the result of a report issued by Hedinger (1915) [see this *Bulletin* Vol. 3, p. 112], who was specially engaged by the Union of South Africa Government to investigate this disease and came to the conclusion that these parasites played some etiological role. Viljoen first gives an exhaustive review of sarcosporidiosis as described in the literature as regards both the symptoms and the lesions which have by some observers been associated with their presence in the muscle fibres. The majority regard the organisms as harmless parasites which are usually found in large numbers in cachectic animals. Apart from Hedinger the only authors who have attributed to them any pathogenic significance are Watson, in a chronic wasting disease—the so-called loco-poisoning of cattle and horses, McGowan, in scrapie in sheep, Roloff in sheep, and some others. It would appear, therefore, from a survey of these observations that if the organisms are capable of producing a diseased condition in animals such a disease would be of a chronic wasting nature.

Hedinger in his investigations found the following changes in lamziekte cattle:—(1) Extensive degeneration of the muscles, consisting of fatty and Zenker's degenerations and brown pigmentation. (2) Alterations in the nervous system especially in the peripheral

nerves. (3) The presence of sarcosporidia in the muscle fibres of all cases. He stated that these parasites could produce severe alterations of the muscle fibres, not only in the fibres in which they were situated but also in those situated in their vicinity, and even some distance away. His principal reasons for associating the parasites with lamziekte were that they were always present in cases of the disease and more frequent than in animals suffering from other diseases.

Viljoen made a study of the sarcosporidia and the muscle changes that might possibly be due to them in 50 animals that had died from lamziekte, and in 50 others that had died from diseases or causes other than lamziekte. Soon after the death of each animal or soon after its destruction samples of muscle were taken from eight muscles from different parts of the body and fixed in 10 per cent. formalin. The samples were cut carefully along the course of the muscle bundles so as to make certain of obtaining good cross sections, which were cut by means of the freezing microtome. The sections were each 12·7 mm. square by 25 microns thick. They were stained with haemalum-eosin and all the sarcosporidia were counted in each of four sections and an average taken. For studying the morbid changes in the muscles longitudinal sections were cut from the same muscles and from the sciatic nerve. At least 12 sections were examined from each muscle and nerve; six were stained with haemalum-eosin and six with haemalum-sudan III.

The cases of lamziekte examined were all in animals that had contracted the disease at the Armoedsvlakte Experimental Station. Sarcosporidia were present in the muscles of 49 out of the 50 animals, the only exception being a young calf four months old. The animals varied in age from four months to 10½ years, but inasmuch as lamziekte rarely attacks very young animals only three cases that were one year old and under were available for examination. The animals were all either in good or fairly good condition. In the majority of cases the sarcosporidia were rare; the average number of sarcocysts per section were as follows:—Masseter 17·9, triceps 9·8, gluteus 9·7, long. dorsi 8·1, splenius 7·8, quadriceps 7·4, psoas 6·9, and supra-spinatus 6·7. In the masseter the parasites were nearly always more numerous than in the other muscles but the relative degree of invasion of the other muscles varied in the different animals.

Nine animals had passed through previous attacks of lamziekte and in none of these were the parasites very numerous. The average was, masseter 19·1, triceps 6·9, gluteus 11·6, long. dorsi 8·1, splenius 6·3, quadriceps 5·4, psoas 5·8, and supra-spinatus 5·5.

In the three young animals, all one year and under, the parasites were completely absent from the muscles of the four months old calf, comparatively rare in the six months old calf, and rare in the yearling.

Hedinger had asserted that young forms of the parasite were more numerous in lamziekte cases. Viljoen found difficulty in drawing a dividing line between the old and the young parasites but an attempt is made in most of his cases to distinguish between the two by including among "young" parasites those which were smaller in size, had no well-developed capsule, and contained rather small sporoblasts, while "old" forms had well developed capsules, contained comparatively large spores, and measured from 45 to 300 microns in thickness and from 100 microns to 1·5 mm.—rarely 2 mm.—

in length. Young forms were those of about 45 microns or less in thickness.

Fatty degeneration was seen in the muscle fibres of 48 out of the 50 cases and was very severe in most of them especially the acute cases of lamziekte. The two cases in which no signs of this degeneration were found were both hyper-acute forms of the disease.

Zenker's degeneration was constantly found in the muscles in all 50 animals. Its severity varied considerably in the different cases being extensive in some and only slight in others. When extensive it resulted in marked destruction of the infected fibres. Very often both fatty and Zenker's degenerations occurred in the same fibres. Atrophy of muscle fibres was only marked in a minority of the cases. Slight localised patches of commencing haemorrhagic myositis were seen in only eight out of the 50 cases.

The connective tissues remained unaltered in the majority of cases but in seven changes were seen in connective tissue cells and sometimes in the formation of fibroblasts. Hyperaemia was well marked in both cases, the blood supply between the fibres being very rich.

No marked alterations could be detected either in the sciatic nerve or in the peripheral nerves situated in the muscle sections. In a fair number of cases the blood supply in the connective tissues of the nerve fibres was very rich and the perineural lymph spaces were enlarged and showed sometimes an increase in the number of endothelial cells. The enlargement of these spaces, however, might have been produced mechanically in most cases during the preparation of the sections. Nerve fibres were normal in most animals and no signs of neuritis were seen. In a few, however, some rare fibres in some nerve bundles appeared to have lost their medullary sheath and to be converted into thin strands in which the neurilemma nuclei were very numerous.

Brown granules as recorded by HEDINGER were observed in some muscles but they were found to be in reality caused by defective staining for fat. HEDINGER also described an interstitial neuritis and other rather marked changes, which Viljoen, however, could observe in no case. Viljoen also regards the so-called funnel forms or Lantermann's clefts described by HEDINGER in the nerve fibres as artificial productions.

It would appear that the sarcocyst exerted no adverse effect whatever by its mere presence upon the invaded muscle fibre; no irritation was set up and no leucocytes were attracted to the parasite. The invaded fibre appeared to be normal in every way, its striations being well preserved and the only notable abnormality was the distension or bulging of the part of the fibre harbouring the parasite. In muscles where pathological changes were present the lesions were evenly distributed amongst the muscle fibres irrespective of the presence or absence of sarcosporidia. In no case were the lesions observed to be confined particularly to the invaded fibres. Special notice was made of some cases which had been on a lamziekte farm for some years and showed a markedly scanty invasion of their muscle fibres with sarcocysts.

Of the 50 animals which the author describes as " non-lamziekte cases, or animals which died from diseases or causes other than lamziekte " 27 died or were slaughtered at the Armoedsvlakte Station and 23 cases were specimens collected and forwarded from different

parts of the country by Government Veterinary Officers, who were for the most part able to make a definite diagnosis of them. They included animals the history of which was, poverty or cachexia 14; killed while in good health 8; suspected lamziekte 7; traumatic pericarditis 3; blackquarter 3; toxaemia, gastro-enteritis and unknown causes 2 each; suspected poisoning, septic metritis, acute indigestion, anthrax, heart-water, tetanus, general oedema, 1 each.

Sarcosporidia were present in 47 of these cases; the only animals the muscles of which did not harbour parasites were a foetus and two calves aged, respectively, two and eight months. The animals varied in age up to 10 years.

The following table gives the average number of sarcocysts found per section in the muscles examined.

Muscles.	Total fifty Cases.	Fourteen Poverty Animals.	Seven Suspected Lamziekte.	Eight Healthy Animals.
Supraspinatus ..	50·2	152·7	6·4	2·2
Triceps	30·3	88·8	6·5	3·1
Masseter	78·1	231·1	5·1	7·7
Splenius	58·3	151	3	4
Psoas	34·2	86·8	1·2	1·1
Long dorsi	17	43·7	4·4	2·3
Gluteus	47·4	168·5	4·8	5
Quadriceps	42·6	130	9·4	3·3

From this table it can be readily seen that the parasites were much more numerous in these 50 cases than in the lamziekte animals. The high average in the non-lamziekte animals is attributed largely to the heavy invasion of the poverty-stricken animals. There was not much difference between the average number in the healthy animals and in the suspected animals.

The enormous increase in the number of parasites in the emaciated animals leads one to believe that these parasites overrun their host in just the same manner as do intestinal parasites when it is in a low state of health and in poor condition. There was no conclusive evidence, however, to show that the sarcosporidia were the actual cause of the emaciation. Three of the animals in which the organisms were very numerous were also infected with intestinal parasites. It is suggested that the emaciated condition of the animals was caused by the intestinal parasites and that the sarcosporidia then had the opportunity of over-running their debilitated host. This view is held to be supported by the figures denoting the muscle invasion in the three cases that had died of traumatic pericarditis (supraspinatus 22·3, triceps 20·6, masseter 16·0, splenius 12·6, psoas 13·0, long. dorsi 15·3, gluteus 13·6, quadriceps 8·3). The parasites therefore were much more numerous than in either the healthy animals or in those suspected to be suffering from lamziekte. Inasmuch as traumatic pericarditis very often runs a slow, chronic course, lasting many weeks, the sarcosporidia, it is believed, are enabled to multiply in the much debilitated animal.

The author next discusses the lesions found in the muscle fibres of the non-lamziekte animals. In the poverty animals the most constant alterations were atrophy of the fibres and an increase in sarcolemma

nuclei. These changes were very severe in some cases. Fatty degeneration, which was very marked in acute lamziekte, was only seen in one emaciated animal. Zenker's degeneration was met with in nine out of the 14 poverty animals although in many cases very few fibres were affected. Changes in the connective tissue were fairly frequent, being present in nine out of 14 cases; these changes comprised an increase in the connective tissue cells sometimes marked proliferation of fibroblasts with formation of new connective tissue in others. Marked myositis, particularly interstitial, was observed in four out of the 14 cases, and was very intense in some animals. In spite of the very high invasion with sarcocysts the muscle lesions in these poverty animals were very slight. Particular cases are noted in which the parasitic invasion was strikingly intense and yet the lesions were quite mild.

The usual degenerative changes seen in lamziekte animals were present in the seven cases of suspected lamziekte, and sarcosporidia were comparatively rare in the muscles of these animals. The eight cases denoted as healthy animals were killed while in good condition and in perfect health. Sarcosporidia were rare in the muscles of all these animals. Five of these, including two that had passed through an attack of lamziekte, were killed at Armoedsvlakte. It could be definitely stated that there was no abnormal increase in the number of sarcocysts in the muscles of the animals that had passed through an attack of the disease. In all five cases a few scanty muscle fibres were seen which had undergone changes very closely resembling Zenker's degeneration, and it is difficult to account for these changes in view of the fact that the animals were in perfect health and condition at the time of slaughter.

With regard to the animals suffering from the other diseases the number of cases of each disease which came under consideration was too small to draw any definite conclusions. Fatty and Zenker's degeneration were not very constantly present. Again the severity of the muscle lesions was entirely unconnected with the degree of parasitic invasion.

The following table shows the average number of sarcosporidia found in nine animals from lamziekte farms and 10 from non-lamziekte farms; poverty cases, suspected lamziekte cases, healthy animals, and two dead from unknown causes are excluded from this list.

Muscles.	Lamziekte Farm.	Non-Lamziekte Farm.
Supraspinatus	8·1	18·4
Triceps	8·9	16·5
Masseter	23·2	11·8
Splenius	5·3	17·1
Psoas	3·5	3·9
Long dorsi	4·5	7·9
Gluteus	7·5	18·3
Quadriceps	6·8	13

These figures show that the average number of sarcosporidia was actually higher in the muscles of animals which had never been on a lamziekte farm.

In the non-lamziekte cases nerve lesions similar to those described in the nerve fibres of the lamziekte cases were seen 15 times out of 50; no special significance could therefore be attached to the appearance of these fibres as the changes were also observed in the nerves of perfectly healthy animals.

[The work done in the course of this investigation has now, it seems, lost a good deal of its point as regards any possible bearing sarcosporidia may have upon the etiology of lamziekte. THEILER and his assistants (including Viljoen) now claim to have discovered the actual cause of the disease, which is a toxin generated by certain anaerobic bacteria which have a localised geographical distribution in the soil and multiply in putrefying carcases; these are ingested by cattle that suffer from a depraved appetite in certain districts in South Africa, and the depraved appetite or pica is claimed to be attributable to a phosphorus deficiency in the food. Viljoen's work, however, is otherwise of great value inasmuch as it provides one with a good deal of precise information upon the much debated question as to the pathogenicity or harmlessness of the sarcosporidia.—ED.]

TYZZER (E. E.) & FABYAN (M.). **Further Studies on "Blackhead" in Turkeys, with Special Reference to Transmission by Inoculation.**—*Jl. Infect. Diseases.* 1920. Sept. Vol. 27. No. 3. pp. 207–239. With 2 plates.

Summary and Conclusions. "A distinct form of blackhead may be produced in turkeys by the subcutaneous inoculation of liver lesions from acute cases, and this disease may be propagated apparently indefinitely, by subinoculation into normal turkeys. This form of the disease is characterized by the appearance of a primary local lesion which first shows appreciable development from 5 to 7 days after inoculation; by the occurrence of secondary lesions regularly in the lungs, rarely in the liver and kidney, and by the later involvement of the liver and kidney (tertiary lesions) through the dissemination of the parasite from the involved lungs. In addition to weakness, loss of appetite, and sulphur colored feces—symptoms which are seen in the spontaneous disease—there is coughing and more or less dyspnea. The inoculated disease has been invariably fatal.

"The incubation period is commonly 11 days, but varies between 11 and 17 days. The appearance of symptoms evidently indicates sufficient involvement of vital organs to interfere seriously with function. The rapid development of the subcutaneous lesion is not attended either by loss of weight or by symptoms, neither of which appears until after the internal organs are involved.

"In the course of their migration through the tissues from the site of inoculation, some of the parasites penetrate the veins and are carried to the lungs where they for the most part lodge and produce lesions. The disease thus metastasizes by way of the blood stream in a manner similar to that of certain tumours. The distribution of the parasites is thus governed by their ability to penetrate vessels, and by their size and physical properties which cause them to lodge in capillaries.

"The expansion and contraction of the lungs evidently serve to dislodge organisms so that these organs are not as effective filters as the liver.

"The parasites develop readily in a variety of tissues and organs: mucous membranes, connective tissues, both smooth and striated muscle, lung, liver, and kidney. Macroscopic lesions of the kidney and microscopic lesions of the lung have been found in spontaneous blackhead.

"The inoculation of chickens has resulted negatively except in newly hatched chicks in which self-limited local lesions, and in one instance, secondary lesions in the lungs, were produced.

"A certain proportion of pigeons have proved susceptible to the extent of developing transient self-limited focal lesions. Positive results were

obtained in 30 per cent. of those inoculated. The lesions are essentially similar to the subcutaneous lesions of the turkey, but after developing actively from the fifth to the eighth day undergo regression. Rabbits, guinea-pigs and mice have proved nonsusceptible.

" Blackhead may be contracted spontaneously from acute cases of its inoculated form, probably from the ingestion of food or water contaminated by discharges from the respiratory tract.

" An attempt to transmit the disease through the agency of a species of ' blow fly ' has failed, but it is possible that this or related species may play a part in the dissemination of the disease.

" Exposure of a young turkey to common fowls, after a long period of isolation, has been followed by the contraction of blackhead—unless the infection is to be attributed to the blackhead-fed flies ingested by it 37 days previously.

" Neither the administration of tartar emetic nor of quinin has served to check the course of the infection.

" Blackhead may be produced by the introduction of organisms beneath the skin, and is not dependent on the invasion of the tissues by myriads of flagellates that have multiplied in the lumen of the gut.

" No flagellates have appeared in the ceca of newly hatched chickens following the ingestion of large amounts of blackhead virus derived from subcutaneous and lung lesions.

" Cases of spontaneous blackhead occur in which there are no demonstrable flagellates in the cecal contents or in the gland lumina.

" The definite transmission of the disease from one turkey to another by inoculation at once removes the great uncertainty which has attended all experiments in the past which have had to depend on the natural and not well understood method of transmission by the exposure of healthy turkeys to supposedly infected turkeys.

" The demonstration of the uniform susceptibility of the normal turkey throughout its period of growth, not only opens the way for further investigation of the question of transmission, but also serves as a basis for future work on other problems relating to the virus of blackhead."

DISEASES DUE TO METAZOAN PARASITES.

DONATIEN (A). **Gale du Dromadaire (3e Note). Essais de traitement par le guetran (goudron arabe) et par le pétrole sulfocarboné.** [Dromedary Mange (3rd Note). Experiments on Treatment by means of Guetran (Arabian Tar), and Petrol with Carbon Bisulphide.]—*Bull. Soc. Path. Exot.* 1920. July 7. Vol. 13. No. 7. pp. 545–548.

A number of camels were brought into Algiers from the high plateaux in the interior for experiments in connection with trypanosomiasis. Inasmuch as they were confined in small paddocks, dry fed, and lived in a humid atmosphere as against the dry climate they had come from they became severely affected with mange, and in such conditions this affection not infrequently ends fatally. From time immemorial the wandering tribes of Algeria have used for preventive and curative treatment against mange a vegetable tar, guetran, obtained by the distillation of *Juniperus phoenicea* and *Thuja articulata*. According to numerous observers this method gives good results but its application has to be carefully carried out otherwise it may result in the death of the animals. The treatment is therefore only carried out by experienced camel men.

The herd of camels at the Pasteur Institute at Algiers was submitted to this form of treatment. In order to prevent re-infection by means of acari scattered about outside the body the soil and other objects with which the camels might come in contact with in the enclosure were very frequently disinfected. The soil was raked over so as to remove the superficial layer of earth and sprayed with a 5 per cent. solution of cresol, and the other objects such as mangers and watering troughs were covered with a 10 per cent. solution of slaked lime. The treatment was successful but was of long duration, lasting over a year, although the treatment was first of all applied weekly, then fortnightly, and finally monthly. At the end of the following summer the disease had almost entirely disappeared, but it again became generalised on all the animals' bodies in November and December. Treatment with tar was then recommenced by smearing with it the whole body once a month. The following June the animals which had not died showed some slight improvement and a female which gave birth to a young camel in February transmitted the disease to its young within the first week after birth.

A few trials were then made with a fluid consisting of one part of carbon bisulphide and three parts of petrol. The animals were scrubbed over with this solution three times at three-day intervals. The lesions showed considerable improvement and the itching was lessened. A female gave birth to a young camel in February, but this young animal developed lesions of mange within three weeks of birth.

The author concludes that in the conditions in which the camels were kept at the Institute, viz., living a sedentary life in a humid climate, guetran proved to be ineffective, at most it only prolonged the life of the animals. The carbon bisulphide solution was a better agent but it failed to eradicate the disease completely.

VELU (H.). **Comment il convient d'organiser la lutte contre la gale des animaux domestiques aux Colonies.** [The Control of Mange among the Domesticated Animals in the Colonies.]—*Bull. Soc. Path. Exot.* 1920. Mar. 10. Vol. 13. No. 3. pp. 186–191.

In spite of the experience obtained during the course of the War opinion is still not unanimous upon the efficacy of any single product for the treatment of mange. However, dips or baths either of liquid or vapour seem to have gained most favour and if their method of employment were more simple their adoption undoubtedly would become universal. No systematic method of investigation was performed with a view to solving this question. In France the only official experiments were performed in connection with sulphur vapour baths. In 1918 the Military Centre of Veterinary Research stated simply that the antipsoric value of arsenious acid and potassium polysulphide has hitherto been greatly exaggerated and for the treatment of a number of animals in dipping baths a solution of cresol and soda (cresol 25, sodium carbonate 10, water 1,000) is the simplest and most efficacious preparation. It would appear, therefore, that there is no method available which can be widely applied in the colonies upon isolated farms and stations.

The author states, however, that outside France and its possessions a number of tests have been made upon the efficacy of sulphur and arsenical baths. For example, in 1915, BEDFORD in South Africa demonstrated that sheep scab could be effectively cured by dipping twice with an interval of nine days in certain solutions. [See this *Bulletin*, 1916. Vol. 4. No. 3. p. 135.] Velu himself states that he has made some tests with one of these preparations, viz., Cooper's Dip, and in the case of the pig obtained complete recovery by dipping four times for about two minutes each time at seven day intervals.

Sarcoptic mange in the horse did not exist in Morocco until the end of the War when it was introduced by horses and mules returning from France. The same preparation enabled the author to eradicate a somewhat widespread outbreak. The animals were placed in a cemented trough with walls 20 cm. high, and a 15 per cent. solution of the product was made and applied by pumping it in the form of a fine jet on to the animal's body. This is preferable to a spray. The liquid after use was collected, filtered through gauze, and used again. In this way 25 animals a day can be treated. It is admitted that the number of animals treated in this way is very small compared with the number that can be treated by passing animals through a dipping tank. The author mentions the arsenical solution recommended by WATKINS-PITCHFORD and the sulphur solution recommended by CHAPIN of the United States Bureau of Animal Industry [this *Bulletin*, 1917. Vol. 5. No. 1. p. 57].

TOVAR (Muñez). **Nota sobre la evolución y biologia de los Boofilidos.** [Note on the Development and Biology of the Boophilidae.]—*El Nuevo Diario*, Caracas, Venezuela 1920. July 26. No. 2717.

A cutting from the above Venezuelan newspaper together with a translation has been received by us from MORGAN (Ed.) Veterinary Surgeon, Puerto Cabello, Venezuela.

The Boophilidae are continuous feeders, that is, they pass the whole of their development from the larval to the sexually mature stages on a single host. After the last moult the adult female continues feeding for four or five weeks, then falls to the ground to lay its eggs and dies. The time taken for the eggs to be laid and hatched varies according to the temperature and humidity of the environment. The hatched out larvae cling to stones and leaves of small shrubs where they lie in wait for a passing host. According to BLANCHARD the males attach themselves to small mammals and terrestrial saurians (lizards, iguanas, chameleons, and tortoises) which they utilise not as a source of nutriment but as vehicles for transporting them in order to join the females. They are not blood-suckers and always remain the same size.

A significant fact claimed to have been discovered by this author is that ticks, or at least the Boophilidae, are endowed with a cutaneous form of respiration as well as a tracheal apparatus, and that they can therefore live under water and develop from larva to adult on a coldblooded host without coming into contact at any period with the atmospheric air.

A small species of crocodile known in Venezuela and Colombia as *baba* was found to have adhering to it at different parts of its body a number of tick larvae, some of which were engorged with blood and

others empty. The reptile was placed in a tank of water about 3 ft. deep, and 9 larvae were found fixed under the hind extremities so that they could not come in any way into direct contact with the atmosphere. After four days five of the larvae were transformed into nymphs, which in due course developed into normal-looking adults possessing the essential morphological features of species belonging to the genus *Boophilus* Curtice 1891.

BEDFORD (G. A. H.). **Anoplura from South African Hosts (Part ii).**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 707–734. With 7 plates.

The following were the various species of lice identified by Bedford on South African hosts :—

Order ANOPLURA Leach.

Sub-order SIPHUNCULATA Meinert.

Genus *Haematopinus* Leach.

1. *H. taurotragi* Cummings. Eland.

Genus *Linognathus* Enderlein.

1. *L. stenopsis* Burmeister. Goats, sheep.
2. *L. tibialis* Piaget. Impala.
3. *L. tibialis* var. *euchore* Waterston. Impala, steenbuck.
4. *L. ungulatus* Piaget. Duiker.

Genus *Linognathoides* Cummings.

Five species recorded from rats, mice, squirrels.

6. *L. faurei* n. sp. Ground squirrel.

Sub-order MALLOPHAGA.

Genus *Menopon* Nitzsch.

1. *M. gallinae* Linné. Domestic fowl.
2. *M. francolinus* n. sp. Crowned francolin, red-necked pheasant.
3. *M. powelli* n. sp. Northern red-necked pheasant, crowned francolin.
4. *M. africanum* Jellog and Paine. Spur-winged goose, domestic ducks, white-backed duck, red-billed teal, white-faced duck, knob-billed duck, S. African sheldrake, moorhen, white-breasted duiker.
5. *M. bucerotis* Kellogg. Trumpeter hornbill.
6. *M. lophocerus* n. sp. Yellow-billed hornbill, red-billed hornbill, South African grey hornbill.
7. *M. impar* var. *poicephalus* n. var. Meyer's parrot.
8. *M.* (*Menacanthus*) *crateropus* n. sp. Pied babbler, Jardine's babbler.

Genus *Colpocephalum* Nitzsch.

1. *C. caudatum* Giebel. White-backed vulture, griffon vulture.
2. *C. subpachygaster* Piaget. Cape barn owl, Cape eagle owl.
3. *C. semicinctum* Rudow. Pied crow.

Genus *Trinoton* Nitzsch.

1. *T. demoratum* Piaget. Greater flamingo.

Genus *Trichodectes* Nitzsch.

1. *T. baculus* Schommer.
2. *T. cuprae* Gurlt.
3. *T. hermsi* Kellogg and Nakayama.
4. *T. limbatus* Gervais.
5. *T. painei.* Kellogg and Nakayama.

} Goats.

6. *T. subrostratus* Nitzsch. Domestic cat.
7. *T. lineatus* n. sp. Steenbuck.

Genus *Goniodes* Nitzsch.

1. *G. aegypticus* Kellogg and Paine. Laughing dove, Damara turtle dove Cape turtle dove, Cape fruit dove. .
2. *G. hilli* n. sp. Red-eyed turtle dove.

Genus *Goniocotes* Burmeister.

1. *G. nigromaculatus* Mjoberg. Domestic fowl, crowned francolin, crowned guinea fowl.

Genus *Philopterus* Nitzsch.

1. *P. gonothorax* Giebel. Southern black-backed gull.
2. *P. cursor* Nitzsch. Giant eagle owl.
3. *P. ceblebrachys* Nitzsch. Giant eagle owl.
4. *P. rostratus* Nitzsch. Cape barn owl.
5. *P. meropis* Denny. White-fronted bee-eater.
6. *P. senegalensis* Rudow. Red-shouldered glossy starling, lesser red-shouldered glossy starling.
7. *P. subflavescens* Geoffrey. Greater puff-back shrike, dusky fly-catcher, Cape sparrow.
8. *P. ornatus* Nitzsch. Black-headed oriole.

Genus *Ibidoecus* Cummings.

1. *I. plataleae* Denny. Spoonbill, white spoonbill.

Genus *Degeeriella* Neumann.

1. *D. actophila* Kellog and Chapman. Curlew sandpiper, little stint.
2. *D. zonaria* Nitzsch. *Tringa subarquata, Tringa minuta.*
3. *D. macrocephala* Waterston. White-fronted sandplover.
4. *D. decipiens* Nitzsch. Avocet.
5. *D. colymbina* Scopoli. White-breasted duiker, grebe.
6. *D. fusca* Nitzsch. Jackal buzzard, sea eagle, gabar goshawk, S. African kestrel, brubru bushshrike.
7. *D. hypoleucum* Nitzsch. European nightjar.
8. *D. erythropteri* Piaget. Little bee-eater, white-fronted bee-eater.
9. *D. argula* Nitzsch. African rook.

Genus *Esthiopterum* Harrison (1916).

1. *E. colius* n. sp. Red-faced mousebird.
2. *E. longicorne* Piaget. White-breasted duiker.
3. *E. fuliginosus* Tashenberg. Cape hen.
4. *E. giganticola* Kellogg. Shy albatross.

Genus *Pectinopygus* Mjoberg.

1. *P. bassanae* O. Fabricius. Cape gannet.

HORNBY (H. E.). **Round-worm Infection of Calves.**—*Rhodesia Agric. Jl.* 1920. Aug. Vol. 17. No. 4. pp. 348–352. With 1 plate.

While acting as Government Veterinary Surgeon in Northern Rhodesia Hornby noted that calves were very liable to heavy infestation with *Ascaris vitulorum* during the first six months of the year.

The presence of these worms in the intestine may be revealed during the other half year but usually it ceases to cause trouble when the mud dries up. This worm is found all over the world but it would appear that there are few places where it does so much harm as it does in South Africa. The full-grown female is 8–12 inches long and the male from

6–10 inches long. The adults are found only in the alimentary tract of calves, for the most part in the small intestine. The fertilised female lays an enormous number of eggs daily, which are passed out with the faeces and under proper conditions of warmth and moisture the eggs were found to contain fully-formed embryos at the end of a fortnight. When they have arrived at this stage they are infective.

The trouble caused by the worms is at its worst in Northern Rhodesia about the month of April. This month is the last of the six warm rainy months, during which a large proportion of eggs have been able to develop fully. As the ground around the calf pens becomes drier and colder an increasingly greater proportion of the eggs fails to develop so that calves born in the winter escape heavy infection.

The author believes that eggs which contain larvae are more resistant than the unsegmented eggs, for one dry season does not appear to suffice to clean infected soil. The opportunities for the multiplication of the parasite in calves after the beginning of the rains are most abundant. The author endeavoured to obtain evidence as to whether the Ascaris migrated into the lungs and various other organs during the early period of its development and there set up lesions on its path. This course has been demonstrated in the case of the pig Ascaris by the recent work of Stewart (F. H.) and Ransom (B. H.) [see this *Bulletin* 1919. Vol. 7. No. 2. p. 73].

The author, however, was unable to demonstrate any changes in the calves which would point to this having taken place. Cases were examined in which the presence of the parasite in considerable numbers in the intestine was found and during life the animals had shown symptoms of coughing and dyspnoea. Lesions of pneumonia were found but no larvae could be seen. Whether the development is accomplished wholly in the intestine or not, it is certain to be very rapid, for dozens of worms ranging in length from 1 to 7 inches were found in the intestine of a two weeks old calf.

The symptoms produced were unthriftiness, loss of appetite, staring coat, pale mucous membranes, dullness, and a tendency towards diarrhoea. The animal blows on the slightest exertion and is liable to attacks of colic. On post-mortem examination the number of worms present in the small intestine is sometimes so great that the bowel is dilated and the outline of worms can be seen readily through the stretched coats.

With regard to treatment the author found that turpentine was the most effective vermifuge. A dose of 2 drachms was found to be safe but ineffective. On the other hand, 1 oz. killed or expelled all worms but was toxic for the calf. The system recommended is to give on two successive mornings from 2 to 4 drachms turpentine in 2 ozs. of a mixture of linseed and castor oils. After an interval of a week a third dose should be given. In order not to disturb milking arrangements the calves are dosed in the early morning and then allowed to run with their mothers.

The effect of Cooper's dip tablets (containing presumably sodium arsenite) was tried. All but the smallest calves could be given three tablets a week for two weeks without showing symptoms of poisoning. A single tablet given on two successive days, then after an interval of a day followed by a third, did not produce any diminution in the number of eggs passed in the faeces after a fortnight. However, there

was some evidence to show that two tablets given at a time once a week for a month will assist in controlling parasitic infestation in a herd. The author insists upon the importance of preventive measures.

BACTERIAL DISEASES.

THEILER (Arnold). **Nodes and Nodules in the Lungs of South African Equines.**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 181–336. With 7 plates comprising 13 figs.

Nodes and nodules are frequently found in the lungs of South African equines and their presence is of particular importance when the animal in question is suspected of glanders and a mallein reaction has to be interpreted. A microscopic survey of such nodes and nodules both from horses affected with and free from glanders was undertaken by Theiler on a very extensive scale, and the results are compared with those obtained in Europe, where this question for a long time was the subject of much controversy.

The latest contribution upon this subject is that of ANGELOFF (1907) who described (1) grey transparent nodules, (2) fibrous nodules, (3) calcareous nodules, (4) glanders nodules, in the lungs of horses. The grey transparent nodules were stated not to be glanders nodules. In the majority of cases they were parasitic nodules, rarely solitary lymphatic nodules. Nodules containing living or dead or degenerated parasites were characterised by the presence of eosinophile leucocytes and could thus be easily distinguished from other nodules. Fibrous nodules might arise from the grey transparent ones but they might also be the product of a chronic catarrhal or proliferating bronchitis or due to an embolus of the blood vessels. The calcareous nodules were caused by the deposition of lime salts in the grey transparent or fibrous nodules. The glanders nodules were regarded as specific products and had to be considered as small foci of hepatisation. The cells which formed the nodules resulting from the action of the glanders toxin underwent karyorrhexis, and the nuclear detritis retained its faculty to take on nuclear staining. Further these nodules did not possess any eosinophiles and did not undergo calcification.

OLT (1910) described (a) grey translucent nodules due to nematode larvae, as originally described by SCHÜTZ; (b) calcareous nodules which had arisen from the above; (c) embolic miliary fibrous nodules situated sub-pleurally or in the depth of the lung tissue consisting of a fibrous tissue enclosing a plug of pin head size which contains a nematode larva. These were rarely found and contained numerous eosinophiles, apparently undergoing calcification. These nodules were connected with a blood vessel. (d) Embolic calcified nodules, which were the final stage of the preceding ones. Theiler quotes at length the detailed description of the structure of glanders nodules which had been given prior to the above by MCFADYEAN, and also refers to the descriptions of the histogenesis of these lesions given in HUTYRA and MAREK's handbook.

As far as the glanders nodule is concerned modern authorities are agreed that it represents a well defined lesion which is typical for the disease, viz., a nodule that is initiated by transudation of fibrin, by emigration of leucocytes, desquamation of epithelial cells, the cellular elements undergoing chromatotexis, and succeeded by a reactive capsule formed of epitheloid cells, fibroblasts and giant cells, which finally produce wavy connective tissue that is connected with the septa of the adjacent alveoli. It is remarkable that none of the modern writers describe healed out glanders nodules, although it is well known that the lesions affecting the mucous membranes of the nose, for example, can heal out.

In this report Theiler deals with nodes and nodules that were collected post-mortem and classifies them under three headings, viz., glanderous, parasitic, and of various origin. The glanderous variety received a great deal of attention and although it is as a rule of a typical structure there were variations found that would appear to be abnormal. All those lesions in which the eosinophile cells were by far the most common cellular elements or at least very frequent ones were regarded as of parasitic origin. In the third group all nodes and nodules were collected that could not be grouped under the above two headings.

In summarising his observations Theiler states that the glanders and the helminthic nodules show histologically an analogous structure and a great similarity, which increases as the pathological changes develop. Both processes begin as a localised pneumonia in which the exudative phenomena prevail. But in the glanders focus it is the neutrophile leucocyte which appears and in the helminthic focus the eosinophile. The greater degree of fibrinous exudation in glanders is probably due to the greater injury produced by the more diffusible glanders toxins upon the vascular system. Whilst a pneumonic focus due to helminthic invasion can be recognised by the presence of eosinophiles in its earliest stages that due to a glanderous infection does not differ from any other bacterial infection so long as the neutrophile cells are intact.

These cells, however, soon undergo changes which lead to a destruction of the nuclei known as chromatotexis, a karyorrhexis process in which the nuclear fragments break up gradually and slowly into particles and finally disappear. In the process of cell accumulation there would also appear to be a difference between the two types of lesion :—In the worm nodule there is a rich collection of eosinophiles so abundant that all lung structure becomes obliterated, whilst in the glanders nodule the normal lung structure is much less obscured and the outlines of the alveoli can be recognised even in that portion of the nodule which is undergoing necrosis. These phenomena may be connected with the functions of the two cell types. The neutrophile cell is believed to act in the first instance chemically by releasing its anti-toxic neutralising substance. The peculiar chromatotectic disintegration may be a phase in this process, for the dispersion of the nuclear detritis would increase the absorbing surface and thus prevent the diffusion of soluble toxin into the periphery.

It is well known that the bacilli are not destroyed in the glanders nodule and their presence can be shown by means of cultures. The

defensive action would thus appear to be directed against the soluble aggressive toxins.

On the other hand, in the case of the worm infestation the defence is apparently intended in the first instance to imprison the parasite, hence the thick collection of cells. Where they come into immediate contact with the body surface of the worm, the cells partially disintegrate, and thus probably substances are released which are intended for a purpose similar to that in glanders, and also for the digestion of the parasite. The toxin not being so diffusible and the digestive enzymes being required centripetally• disintegration of the cells is limited to the immediate surroundings.

The first object of cell emigration apparently is the seclusion of the injurious substance. Then in addition the vascular and connective tissue react so long as the stimulus is present. The inflammatory process, however, recedes as soon as the necrosis of the affected elements has become pronounced. Another process, namely, organisation then manifests itself. In both types of infection it proceeds along similar lines, viz., from the production of fibroblastic tissue to that of fibrous tissue. In this process epithelioid cells and giant cells appear, which are more frequently found in glanders than in worm infections. These cells have apparently all the same origin. The process appears to represent and is still intended as a defensive action towards the toxic influence since these cells collect mainly in the inner periphery of the developing capsule and centripetally undergo necrosis. In glanders the process continues for a long time as the bacteria in the centre do not die off ; in the worm infection it comes to a standstill sooner as the parasite dies off or otherwise disappears. In the worm nodules the inner periphery of the epitheloid cell zone is observed to undergo necrosis but to a much smaller extent than in glanders. The necrosis is probably due to the escape of toxin during the disintegration of the parasite. Once the injury is removed permanently the final organisation apparently proceeds fairly quickly and leads to the formation of the fibrillate and hyaline capsule, which in turn disappears until only a centre is left apparently destined to be finally calcified. In glanders the final stages were not recognised but one nodule was observed to calcify in the centre whilst the epitheloid cell zone was still present.

But Theiler sees no reason why the process should take a different course in the two types of lesion once the toxin-producing bacteria are finally removed, for the presence of the necrotic central substance acting as a foreign body on the connective tissue must of necessity lead to its occlusion. An actual deposition of lime salts, however, as was noted in one observation, must be the usual ultimate issue. But this process seems, however, to be a rare one.

Parasitic and glanders nodules resemble each other in the fibrous organisation and the older the process the greater the similarity. But so long as eosinophiles are present a distinguishing factor is present. Even when these have disappeared the origin of a nodule can be recognised for a long time by the pink or dark staining of the central mass which in the parasitic nodule remains for a very long period. In glanders the reverse takes place, the central mass tends to become paler and fades in the process of further organisation while the nuclear substance breaks up into fine granules which finally disappear. In

parasitic nodules the nuclei become pycnotic together with the eosinophile substance, which apparently is of a persistent nature, and tends to remain pink or dark pink.

In the numerous examinations undertaken no nodules were seen in which there might have been any doubt as to their origin except those in which the centre was fully calcified, and in which this difference of shade in the necrotic substance could be no longer observed.

Macroscopic Aspect of the Nodules and Differential Diagnosis.

Glanders Nodule. This begins as a miliary pneumonia with well marked exudation, consisting of polymorphonuclear neutrophiles and liquid plasma. The plasma coagulates, fibrin is deposited, and the separating serum soaks through the whole structure. These phenomena are preceded by a vascular disturbance and thus at the outset one would expect to find on naked eye examination a hyperaemia, which later, however, becomes limited to the periphery. A small nodule appears at the place where the leucocytes have collected and this point remains translucent as long as these cells are intact. The cells, however, soon disintegrate, a deposit of fibrin is formed and then this part ceases to be transparent. The translucent early stage is thus a very short one, and very rarely encountered, except in experimentally produced cases.

The fibrinous miliary focus appears in section as a greyish reddish area with somewhat irregular outlines; its cut surface is slightly granular and protruding whilst the surrounding periphery is smooth, gelatinous, and injected, as the alveoli are filled with oedematous liquid and desquamated cells and the capillaries are engorged with blood.

A somewhat later stage has a different naked-eye appearance. Disintegration of the leucocytes and the formation of necrotic substance leads to an increase in opacity of the centre which becomes dirty white, in contrast to the greyish reddish gelatinous looking periphery. The relative size of these two zones varies in different nodules and sometimes even in the same nodule. The transition from the outer zone to the centre is by no means well defined although the most central part stands out distinctly as a white pith which now represents a small abscess. This has a soft consistence and it is frequently removed by the knife in cutting into the nodule.

Multiplication of the fibroblasts now takes place and forms a transition stage. A zone around the necrotic white centre becomes more and more well defined as a greyish-white dense smooth, sometimes transparent, capsule with a tendency to become more and more white towards the periphery. The periphery, however, is not well circumscribed, as fibrillar strands run into the adjacent septa, so that the structure cannot be lifted out with the knife. The pith, which is more or less liquid, subsequently becomes inspissated and dirty white to yellow in colour and somewhat sticky. The fibrillar tissue increases in quantity and the capsular nature of the nodule becomes more and more pronounced. The peripheral hyperaemic zone gradually disappears.

Theiler observed no stages beyond the above but believes that the nodules then become identical with the nodules produced by worms, except that the peripheral arrangement of the fibrillae might be different. Only one calcified nodule was observed, which had not reached the latest stage in the development of the lesion.

Variations sometimes occur in the evolution of the lesions as summarised above. In the earlier stages the presence of blood sometimes changes the picture. The injected blood vessels may persist for a longer or shorter time in the central portion or haemorrhages may occur within it. It will therefore be more reddish in colour and patchy in appearance. The capsule when it consists of young fibroblasts is more or less translucent. When the pith is very small the whole nodule may appear translucent. Sometimes the nodules appear as suppurating granular tissue, impossible to distinguish from any other such tissue. The presence of typical glanders nodules alongside these abnormal ones may serve as a guide for diagnosis. These lesions appear more opaque and diffuse greyish-white, with irregular outlines, and sometimes as broken down suppurating ulcers.

Worm Nodule. This nodule shows greater naked-eye differences than does the glanders nodule, and this is due to the fact that most of the worm nodules originate in the bronchi. The typical nodule is translucent, for the eosinophile cells remain intact for a much longer period than do the neutrophiles. The nodules only undergo necrosis in the centre and the centres appear as well defined white specks which have varying shapes and sizes depending upon their situation—whether in the lumen of a bronchus or in the alveolar tissue. They are frequently irregular in outline and usually stretched in the longitudinal direction. Their consistency is not so soft as that of the glanders nodule and it may be described as putty-like. Translucency remains for a considerable time even while connective tissue formation is taking place.

Subsequently the nodule gradually becomes white, and a definite capsule is formed which is fairly well circumscribed both outwardly and inwardly. The nodule develops the character of a fibrous nodule more and more markedly as the lesion progresses in age. The central pith increases in size, assumes more rounded contours, has a dirty whitish-yellow appearance, and contains fine granules. It protrudes on the cut surface and it is crumbly, and often contains calcareous and hard spicules. The capsule becomes thick, hard, and horn-like in consistency.

Finally, only a thin capsule is left on account of the increase in size of the necrotic centre. The capsule is well separated off from the adjacent alveolar tissue so that the nodule can be lifted or squeezed out. The pith is calcareous, brittle, or hard, and resembles a small marble as it can no longer be cut.

Difficulties may occur in naked-eye diagnosis during the younger stages when phenomena other than eosinophile emigration supervene,—for example, hyperaemia and haemorrhage, exudation of fibrin and the presence of a transudate which permeates the adjacent tissue, desquamation of the alveolar epithelial lining distribution of cells in the different parts of the tissue and their arrangement in clusters or streaks and irregular distribution of fibroblastic and fibrillar structure. These various factors contribute to the formation of a smaller or larger focus with an irregular outline and a different colour and consistency, and the nodule appears as a pale yellow, fleshy or fibrous structure. The lesion due to worm invasion is invariably a translucent grey nodule in one of its earliest stages and a white fibrous and calcareous structure in its later and latest stages. Typical worm nodules may be mistaken for very small lymph nodules but these, however, are not frequently encountered. These lymph nodules possess a thin fibrillar capsule.

In the fibrous stage a parasitic nodule may be mistaken for a glanders nodule but the difference in the consistency and in the appearance of the central pith, and the ill-defined outer periphery serve as aids in arriving at a diagnosis.

Nodules not due to Glanders or Parasites. These have no definite naked-eye appearances and appear as haemorrhages, consolidations, abscesses, or sequesters and lead to the formation of cicatricial tissue. They are not always well-defined. Nevertheless these structures may cause doubt in one's mind, for glanders may also take on an atypical course and lead to the formation of a suppurating granulation tissue and a fibrous structure (malleosis occalescens.).

Whilst, therefore, there are sufficient points to enable one to distinguish between the different types of nodules macroscopically in the majority of cases, there are some atypically built lesions in which the origin of the lesion cannot be recognised without histological examination.

Mallein Reaction and Diagnosis of Glanders. A study of the case records showed that there had been in several cases a discrepancy between the diagnosis arrived at as the result of the mallein reaction (subcutaneous and intra-palpebral) or the complement fixation and agglutination test on the one hand and the result obtained upon post-mortem examination on the other. The number of cases in which this occurred would lead one to believe that the methods of diagnosis employed were not reliable. Experience, however, has shown that any one or all of these tests when employed systematically affords an almost certain means of detecting glanders.

Theiler attempts to explain away the discrepancies as follows :— Where the diagnosis during life had been positive and after death negative the most likely explanation would be that not all the lesions were detected on post-mortem examination ; inasmuch as glanders lesions may be present in organs other than the lungs they may be frequently difficult to find. Again, no discrimination was made in the selection of the nodules for histological examination so that very likely glanders lesions were sometimes missed. In several cases, however, particular care was taken to collect all the nodules present and yet glanders nodules were not found amongst them.

The positive results obtained by means of the complement fixation and agglutination test after the test with mallein had proved negative was probably due to the appearance of specific antibodies in the blood as a result of its responding to the mallein as an antigen. Where the post-mortem result was positive, after a negative clinical diagnosis the only explanation that can be given is that the animals were at the time of the test passing through the incubation period of the disease and that the time had not been reached when an allergic reaction could be obtained.

However, taking all things into consideration it has to be stated that mallein testing or any other diagnostic method occasionally leads to doubtful and erroneous results, not so much in that they fail to trace infected horses but in that they produce what have to be termed reactions in healthy horses. However, it is wise and economical to proceed to adopt these tests in order to eradicate the disease even though a number of the horses that do react may not be infected with glanders.

ROBINSON (E. M.). **The Value of the Complement Fixation Test in the Routine Diagnosis of Glanders.**—*Union of South Africa, Dept. of Agriculture, 7th & 8th. Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 579–584.

In this short article Robinson describes the results obtained by the application of complement fixation test for the diagnosis of glanders in horses admitted to the Onderstepoort Laboratory for experimental purposes. The test was carried out in conjunction with the subcutaneous mallein test from June 1914 to January 1916, when it was discontinued on account of the inaccuracy of the results obtained. Such results were obtained in particular among horses that had been used for transport during the German South West Africa Campaign, and had been tested by means of the mallein test, sometimes several times in succession. In all 1,400 tests were made, 240 of which were re-tests. The sero-diagnosis of glanders had previously been performed at the Institute by MEYER (F.F.) 1909–1910. This worker claimed to have obtained good results by using the complement fixation and agglutination tests in conjunction.

Robinson used the method described fully in *Bulletin* No. 136, 1911, of the United States Bureau of Animal Industry. All sera were inactivated by heating to 62–63°C, and very few non-specific fixations occurred; only very occasionally did spontaneous coagulation take place at this temperature. Extracts of cultures, shaken and centrifuged, were always used as antigens. A most successful method of obtaining haemolytic amboceptor is stated to be that described by COCA (A. F.) 1916. Rabbits were inoculated intravenously with 1 to 2 c.c. of washed sheep's corpuscles, and, after an interval of four days, the same quantity was again injected intravenously. Six to seven days after the second inoculation the rabbits gave sera with high titres, up to 1–4,000. Guinea-pig serum was used as complement; occasionally the serum of a particular guinea-pig was found to be quite useless on account of its low complement content.

A few conglutination tests were made and the results were found to correspond roughly with those obtained by the complement fixation test. The agglutination test was used as a control in some cases but great difficulty was encountered in getting an agglutinating strain of the glanders bacillus as it usually lost its agglutinability after having been sub-cultured a few times. Robinson divides his cases up into six groups :—

(1) Negative to complement fixation and not glandered. Of these 1,042 out of a total of 1,160 were obtained. Diagnosis was confirmed later at post-mortem in 756 cases.

(2) Positive to complement fixation and glandered. Of these 15 cases were encountered. In all of these with the exception of one horse a positive complement fixation test and a typical mallein reaction was obtained. The exceptional case gave a temperature reaction only to mallein.

(3) Glandered but negative to complement fixation. Four cases occurred, all belonging to one batch and all acute cases. One gave a temperature reaction and a small suspicious local reaction to mallein

and died a week later from glanderous pneumonia. The other three developed farcy within a fortnight of purchase and were destroyed.

(4) Positive to complement fixation and mallein, but not glandered. Of these, six cases occurred. One gave a temperature reaction and a very slight local reaction to mallein and subsequently the mallein tests on it were atypical or apparently negative. It, however, continued to give positive results to the complement test. On post-mortem a few fibrous nodules were found in the lungs, but inoculation of guinea-pigs with material gave negative results. A second horse first gave a typical mallein reaction but a month later failed to react. At this time the complement fixation result was positive but it subsequently gradually declined. The four other horses gave results that were undoubtedly positive to the complement fixation test on account of their having been subjected to injections of mallein previously; the anti-bodies were in all cases lost after the mallein injections had ceased.

(5) Seven horses gave reactions to mallein, most of them being not decidedly typical but strongly suspicious. The complement fixation test in all these was negative. In three of these which gave typical temperature reactions but not typical local reactions glanders was not detectable post-mortem. One showed post-mortem a few nodules, the nature of which was not determined, but they were probably not due to glanders. One gave at first a negative mallein reaction, but a month later a temperature and a fairly good local reaction; post-mortem result was negative. The remaining two horses gave indefinite reactions, both temperature and local, and were negative post-mortem.

(6) A large number of cases, 86, were obtained of horses which gave a positive complement fixation reaction on arrival and were negative to mallein (by positive reaction is meant complete fixation with quantities of 0·2 and 0·1 c.c. of serum). Some cases were found to be positive with quantities of 0·01 c.c. of serum and were not glandered. Sixty-two gave complete fixation and 24 partial or incomplete fixation, in most cases absent when quantities lower than 0·1 c.c. of serum were used.

Sixty-six out of the 86 cases were horses purchased from the Defence Force or Police, and probably all of them lost their glanders antibodies within two or three months. A few horses which on arrival gave negative complement reactions developed positive ones later but nearly always previous injections with mallein could be put down as the cause. At the close of the Campaign a large amount of mallein testing was done upon the horses and many had been injected several times within a few months.

These results therefore indicate that in nearly all cases where a positive reaction to the complement fixation test is obtained in a horse that is not glandered the reaction may be attributed to a previous injection with mallein.

"The main point which this paper is intended to bring out is the unreliability of positive reactions to complement fixation in horses that have previously been malleined, even where a month has elapsed since the last mallein test, unless that test was the first and not the last of a series."

VELU (H.). **Quelques observations relatives aux pasteurelloses au Maroc.**—*Bull. Soc. Path. Exot.* 1920. July 7. Vol. 13. No. 7. pp. 493–497.

In this short note the author calls attention to the affections caused by organisms of the fowl cholera or haemorrhagic septicaemic group in Morocco.

The animal chiefly affected with these organisms appears to be the pig, and a heavy mortality is sometimes caused by diseases due to the same type of organism also in other animals. In pigs the infection is often mixed but sometimes it nevertheless manifests itself in a pure form and causes very high mortality. The form which it usually takes is the oedematous form shown by acute general symptoms together with the appearance in the region of the throat of a soft oedematous swelling. Young animals are much more susceptible than adults. The disease is seasonal and occurs chiefly in the spring. Every year outbreaks are reported almost simultaneously in different districts.

Diagnosis is usually very difficult inasmuch as the condition may be easily confounded with anthrax and the nature of the disease can only be identified by laboratory examination. On post-mortem examination one finds in the swollen parts areas of blood-stained infiltration from which exudes a sero-fibrinous lemon-coloured or brownish fluid; the muscles usually show minute haemorrhages; the lymphatic glands are soft on section especially in the oedematous regions. The outbreaks always run a characteristic course. They are sudden in their onset and cases of death rapidly follow each other and then as a rule the disease disappears just as suddenly as it appeared. These occurrences are similar to what takes place in fowl cholera.

One outbreak of haemorrhagic septicaemia among cattle presented peculiar features; calves alone were attacked and at first it was thought they were affected with an acute attack of white scour. Symptoms were shown suddenly; the animals became dull and lost their appetite; and then they became affected with acute colic and severe straining; the faeces were at first normal and covered with mucus or blood, but later they became diarrhoeic. Death occurred usually in 12–24 hours. Some cross-bred Moroccan zebus survived for two or three days. Two adult animals, a working ox and a milch cow, were attacked and succumbed. Apart from symptoms in connection with the digestive tract they presented characteristic swellings about the head and tongue. In the calves also there were signs of dysphagia and salivation. The disease was present in the young pigs of the establishment and it ran the usual characteristic oedematous form. The organisms were isolated in culture and by the inoculation of pigeons. The origin of the outbreak was traced to the drinking water. As soon as a diagnosis was made the animals each received from 5 to 10 c.c. of anti-serum and the disease then immediately disappeared. A calf left uninoculated, on the other hand, died with the usual symptoms. The serum was prepared by using various strains of the organism isolated from the pig and of the fowl cholera organism.

VERGE (Jean). **La pasteurellose du buffle en Syrie.** [Haemorrhagic Septicaemia of the Buffalo in Syria.]—*Bull. Soc. Cent. Méd. Vét.* 1919. Dec. 18. *Rec. Méd. Vét.* 1920. Jan. 30. Vol. 96. No. 2. pp. 52–55.

In certain parts of Syria where buffaloes are kept in very large numbers haemorrhagic septicaemia does not appear to exist, but in the Houlai Plains and along the course of the Jordan, where buffaloes are used by certain Bedouin tribes in the district to replace oxen in farm work, the disease exists as a very deadly enzootic which causes great ravages among the herds in certain years. The disease is seasonal and appears towards the end of winter and rages throughout the springtime. The disease again disappears in the winter. It is believed by the natives that rinderpest and haemorrhagic septicaemia never occur simultaneously among the herds of buffaloes. Adult and very young buffaloes seem to be most resistant and animals from six months to two or three years old suffer most heavily. The author describes fully the symptoms of the disease, which would appear to be similar to those recorded elsewhere. The disease runs its course in from 6 to 24 hours as a rule. Unfortunately, the author was not able to perform any bacteriological examination.

MEYER (K. F.) & SHAW (E. B.). i. **A Comparison of the Morphologic, Cultural and Biochemical Characteristics of** *B. abortus* **and** *B. melitensis.* **Studies on the Genus** *Brucella* **Nov. Gen. I.**—*Jl. Infect. Diseases.* 1920. Sept. Vol. 27. No. 3. pp. 173–184. With 8 figs.

—— & FEUSIER (M. L.). ii. **Principles in Serologic Grouping of** *B. abortus* **and** *B. melitensis.* **Correlation between Absorption and Agglutination Tests. Studies on the Genus** *Brucella* **Nov. Gen. ii.**—*Ibid.* pp. 185–206. With 14 charts.

In these two papers a series of laboratory experiments are described which are held to indicate that there is a very close relationship between the causal organisms of the common form of contagious abortion in cattle and of Malta, or undulant, fever in man. This relationship was first suggested by some work published by EVANS (Alice E.) in 1918, although serious attention was not paid to this author's work at the time owing to the fact that the organisms were also supposed to bear some resemblance to *Bacillus bronchisepticus*, which is obviously a different type of organism. The term micrococcus as applied to the Malta fever bacterium is strongly disputed.

In the first paper a comparative study is described of 21 cultures of "*Micrococcus*" *melitensis* obtained from various parts of the world and of 32 cultures of *B. abortus* Bang isolated in U.S.A. and Gt. Britain. It is held that the two organisms cannot be distinguished morphologically or biochemically. The so-called "*Micrococcus*" *melitensis* appears in young cultures as a short rod and ought therefore to be designated as *Bacterium melitense.* The pigment production of the majority of the actively growing Malta fever strains on a medium called glycerol-peptic-digest agar and on alkaline potato cylinders after five days incubation was more intense than with the strains of *B. abortus.* Cultures of both organisms produced "after 20 days

incubation in glucose and lactose broth an alkaline reaction and a characteristic reduction of the H-ion concentration equal to about 0·6 to 1·0 PH."

In the second paper a number of cross-agglutination or absorption tests were carried out on 14 strains of the abortion and Malta fever organisms. The anti-sera used for the test were obtained by immunising rabbits with both dead and living cultures of the organisms. In some of the tests monkey and guinea-pig anti-sera were also used. The organisms were suspended in weak formalin solutions for the tests. It was concluded that the 14 strains could be divided serologically into four groups; Group 1 contained one Malta fever and one abortion strain, Group 2 contained nine Malta fever strains, Group 3 contained one Malta fever strain, and Group 4 two para-melitensis strains. However, all other abortion strains tested were found to fall into Group 1. There was, however, a close relationship between Groups 1 and 2, which were sharply defined from Groups 3 and 4.

In the course of this work the authors claim to have established three principles, viz.: (1) That an anti-serum cannot be exhausted by strains of another group. It is always exhausted by its homologous strain and may be exhausted by other members of the same group. (2) That a strain acts in a uniform manner (qualitatively) on all strains in another group under the same absorption conditions. This uniform action constitutes the basis for group affiliation. (3) That strains within the same group do not necessarily act in a uniform manner on one another when absorbed from the same anti-serum. This constitutes the basis for individual differentiation.

It is suggested that *B. abortus* and *B. melitensis* ought to be given generic rank in the Bacteriaceae family as the genus "Brucella," in conformity with the system of classification adopted by the Society of American Bacteriologists. A series of agglutination tests with the two types of organism revealed a gradation in the highest limit of agglutinability for the different strains. These gradations were constant for each strain when used against the various anti-sera.

The sera of cows and pigs suffering from naturally contracted contagious abortion was found capable of reacting to both *B. abortus* and *B. melitensis*.

[The problem presented in these papers is, on the surface, one of most serious import. Are the organisms of contagious abortion in cattle and Malta fever in man identical or so closely related that they cannot be differentiated except by delicate laboratory tests? A perusal of these papers would incline one to think that that was the case, but the conclusions are couched in somewhat vague and diffuse terms, and the real question of importance is not directly attacked. The fact is that only one of the Malta fever strains fell into the same group as the abortion strain. Nearly all the rest fell into a group by themselves. Besides, there can be but little question as to the absence of pathogenic relationship between these organisms, for while in this country contagious abortion in cattle is rampant and no precautions are taken to exclude the contamination of human beings by milk, Malta fever, on the other hand, happily does not occur.—ED.]

DISEASES DUE TO FILTERABLE VIRUSES.

DE KOCK (G. v. d. W.). **Further Observations on the Disease Equine Pernicious Anaemia.**—*Union of South Africa, Dept. of Agriculture 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 587–636. With 13 tables.

Towards the end of 1913 equine pernicious or infectious anaemia was for the first time recognised in South Africa by THEILER and KEHOE [see this *Bulletin*, 1916, Vol. 4, No. 3, p. 120].

After showing that it was the same disease as occurs in Europe, America, and Japan, these authors called attention to its great economical importance insofar as it affected the immunisation of horses against horse sickness. Further experience has shown that it is very difficult to diagnose the disease definitely when once it has made its appearance, or when it is suspected during a horse-sickness immunisation process. Remissions and exacerbations of temperature often occurred in some of the injected horses and frequently these reactions could not be accounted for clinically; other symptoms such as dirty conjunctival mucous membranes, with petechiae, dullness, loss of appetite and other symptoms were observed in several cases. Negative results were always obtained upon testing these animals by blood examinations and blood injections. After one attack they usually recovered completely. It is suggested that some of the so-called poverty or debility cases for which clinically no cause could be assigned might in reality be stages of infectious anaemia. Frequently swellings appeared on these horses without any apparent cause and then disappeared after a few days. Some animals, after immunisation, did not thrive for some unaccountable reason. Finally, the only method of diagnosis which is capable of furnishing satisfactory information, i.e., blood inoculation, could not always be relied upon especially when the result of the test turned out to be negative.

Hitherto no natural outbreaks of the disease such as have been reported to occur in Japan, America, and other countries have been observed by the Veterinary Division in South Africa. Information with regard to the distribution of the disease was therefore only obtainable from a few reports sent in from districts and dependence was placed mainly on investigations made at Onderstepoort, where the majority of horses utilised for experimental purposes were unsound and, in many cases, decrepit animals, purchased indirectly from different centres in the Union, but mostly from the large towns. An attempt is made to select as far as possible animals which are to serve for the production of virus or serum in horse sickness experiments, avoiding horses with swollen limbs, dirty conjunctival mucous membranes, and irregular temperatures.

Since 1915, 650 horses had been hyper-immunised for the production of horse sickness serum and 130 selected horses were utilised in order to furnish the necessary virus for their inoculation. Fortunately, in this manner no single case of infectious anaemia was directly produced in any of the serum horses. It would thus appear that the disease is not so very prevalent in South Africa as was at one time suspected. In July 1914, however, the introduction of carriers had been definitely ascertained. The distribution of the disease is most difficult to estimate

especially when it assumes the sub-acute or chronic forms, as it is then often mistaken for biliary fever, and the two conditions may be easily mistaken for each other post-mortem.

Many so-called attacks of biliary fever are claimed to have been successfully treated in practice with trypan blue and other drugs; most probably these were cases of infectious anaemia, as it was conclusively shown in the Laboratory that all cases of biliary fever that were definitely established as such by microscopic examination beforehand failed completely to respond to drug treatment.

In Durban, Natal, an outbreak of infectious anaemia was mistaken for dunziekte (see p. 331). Some of the hyper-acute forms of infectious anaemia are easily mistaken for some forms of horse sickness, even upon post-mortem examination. Enlargement of the spleen and acute heart lesions may be common to both diseases. With the frequent occurrence of staggers (acute liver atrophy) [this *Bulletin*, 1920, Vol. 8, No. 1, p. 63], amongst horses immunised against horse sickness since 1916 specimens were very often forwarded to the Laboratory from various centres in the Transvaal for the diagnosis of this condition. In many cases no lesions of liver atrophy were present but the changes were very suspicious of pernicious anaemia, and De Kock's own observations in the Laboratory showed that horses in the last stage of infectious anaemia may show symptoms having a close resemblance to those of acute liver atrophy. The presence of carriers often only becomes apparent after the disease makes its appearance in animals inoculated with the blood of such carriers. It would be very costly and impracticable to ascertain the infectivity of the blood of these animals by blood inoculation on account of the large number that would be required.

There is no doubt but that the disease occurred in South Africa prior to 1914 when it was first definitely diagnosed, and from an examination of the Laboratory records suspicious cases appear as far back as 1904 and thence forward there seem to have been quite a number of cases. During the period 1904–1917 records in connection with 8,906 admitted are available. Amongst these there were only four that could be regarded as positive cases of the disease (i.e., less than 0·5 per cent.) and 72 doubtful cases. There were 33 which could be classified as infectious anaemia after they had been subjected to treatment at the Laboratory and in addition 79 doubtful cases. The diagnosis of these cases was based on the temperature and other observations made during their stay at the laboratory.

De Kock proceeds to analyse the history of these cases in detail. It transpires from such an examination that there does occur a considerable number of secondary reactions following upon the inoculations involved in the production of horse sickness immunity (post-horse sickness reactions, which usually occur a few days and sometimes several days after an animal has passed through a typical horse sickness fever reaction, reactions after hyper-immunisation, and bleeding). These reactions might be easily mistaken for infectious anaemia. It was often impossible to incriminate definitely any particular virus or serum as the source of infection in most of the cases that occurred after treatment.

In order to exclude contamination with the virus of infectious anaemia from the material used for the immunising of horses against

horse sickness it is suggested that the following plan might be adopted in the preparations of the "virus" until some simpler method of diagnosis has been devised :—(a) Select for "bleeders" fairly young horses in good condition ; keep them under observation for at least a month ; if no symptoms suspicious of infectious anaemia are observed they can be utilised. (b) Before they are injected with the horse sickness virus test their blood by injecting it into at least three other horses. (c) After virus has been taken from these horses test it again on at least three susceptible horses in order to ascertain its horse sickness virulency and simultaneously whether it is free from infectious anaemia. If the virus prepared is of the virulent horse sickness type instead of the attenuated type this test must be carried out on immune horses instead of on susceptible ones.

In order to exclude contamination with the virus of infectious anaemia from the anti-serum to be used in the immunising of horses against horse sickness all serum should be stored for at least one year. Storing for three years did not cause any depreciation in the value of the serum. Heating, on the other hand, did not seem to be effective. Serum infected with infectious anaemia was heated for 20 hours at 45° C without destroying the virus.

In addition to the symptoms described by THEILER and KEHOE in 1915 de Kock noted bleeding from the nostrils and skin and sometimes symptoms of so-called "staggers" in a few acute cases prior to death. A careful enquiry was made into the state of the bone marrow in animals that had died from infectious anaemia and many other diseases. Pathognomonic lesions in this situation have been described by some authors. Confirmation was again obtained of THEILER and KEHOE's conclusions "that the results they obtained do not allow them to regard the bone marrow of animals dying from infectious anaemia as presenting any special characteristic appearance, since exactly similar appearances are to be met with in animals coming to autopsy as a result of death from a large variety of causes."

Experiments were undertaken in order to ascertain whether a serum could be prepared from animals clinically recovered from pernicious anaemia that might have curative or preventive properties. No such properties were demonstrated, but, on the contrary, some of the inoculated horses developed the disease. A number of interesting facts were brought out from the experiments.

(a) The serum did not yield uniform results when injected into susceptible animals and only a certain percentage of inoculated horses subsequently showed symptoms of the disease. Serum taken on one particular date from an infected horse did not always reproduce the disease and similarly serum taken on different dates did not invariably produce the disease.

(b) Infective serum was found to be still virulent after having been kept for six months. The infectivity of blood from infected animals appeared to be more uniform than that of serum when injected in fairly large doses to susceptible animals, but here again the results were not constant.

There is no explanation forthcoming as to why the virus present in the blood or serum is capable of producing the disease while it fails in others, which are not virus carriers.

It would appear from a number of observations that there do not exist any variations in virulence of a particular strain or any difference in the virulence of different strains in infectious anaemia. It was not found possible by re-inoculating known virus carriers that had clinically recovered to produce in them the disease "afresh," or cause a "relapse" or "break-down in the immunity."

Serum prepared from clinically-recovered horses that had been subjected to inoculation with massive quantities of virulent blood acquired no therapeutic properties.

De Kock demonstrates in tabular form the results of attempts at curative treatment by means of a variety of drugs, including arsenophenylglycine, trypan blue, camphor, brilliant green, malachite green, turpentine, methylene blue, arrhenal, thymol, creosote, eucalyptus, quinine bihydrobromide, carbolic acid, and alcohol, given singly and in various conbinations.

After the employment of certain drugs, especially arsenophenylglycine, fresh remissions of temperature or relapses were caused in horses, some of which were regarded as clinically recovered. It is suggested that an experiment ought to be carried out on a large number of horses in order to ascertain whether this reaction might be employed as a method of diagnosis for clinically recovered virus carriers. Otherwise the drugs used did not appear to exercise any appreciable effect on the course of the disease.

To exclude the possibility of spreading the infectious anaemia virus during the course of immunising against horse sickness the author now adopts a fresh set of sterilised instruments for each animal.

THEILER (Arnold). **Paralysis of the Oesophagus in the Horse as a Sequel to Horse-Sickness.**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 339–357.

In the course of experiments performed in connection with the immunisation of horses against horse-sickness, symptoms were on several occasions noted that corresponded in every respect with those of the few cases of paralysis of the oesophagus already described in veterinary literature. It would appear that this condition is not uncommon as a sequel to the dikkop form of horse-sickness. It occurred among horses both at the Laboratory and in the field, but very few cases occurred amongst mules, probably because dikkop is usually much less severe in these animals. The disease was noted in inoculated animals only, but it seems that the condition is also set up in horses and mules which contract the disease naturally.

Fifteen of these cases are described in detail and all had been injected with a horse-sickness virus and a reaction had followed in the usual manner although it varied somewhat in length in the various horses; but, it was generally of a mild type. The first observations dated back to 1908, but lately the number of cases has been greater, corresponding with an increase in the number of horses submitted to the immunisation process. The history in some cases, however, would lead one to suspect the particular strain of virus employed. The dikkop swelling usually appears towards the end of the fever reaction and its intensity varies considerably in different animals. It was after the onset of the

typical swellings that paralysis of the oesophagus occurred. In nine cases the paralysis was noticed in dikkop which appeared on or before the 13th day, and in five cases on or after the 14th day. It also occurred in one hyperimmunised horse that developed dikkop on the 15th day. Usually the paralysis manifested itself from three to eight days after the onset of dikkop, and it was longest in appearing in those cases which developed dikkop early. It thus apparently represented a sequel of a severe form of dikkop.

The symptoms were the same as those usually associated with the condition : presence of ingesta in the nostrils, coughing, swellings of the throat, increased thirst, and regurgitation of water after drinking and feed material after deglutition, later offensive odour from the nostrils together with a muco-purulent discharge. Recovery took place in two cases, in the first in one day and in the second in three days. The others became complicated with bronchitis and pneumonia. Some of the horses were killed at an early stage.

Post-mortem. There was an impaction of the oesophagus in all but one case : sausage-like masses of well-masticated and tightly compressed food fell out on slitting up the oesophagus.

Theiler discusses the pathogenesis of the condition and concludes that it is a paralysis of the oesophagus which occurs independently of that of the pharynx and is caused by an interference with the afferent nerves from the pharynx. There nerves ordinarily are supposed to carry the stimulus which travels ultimately through to the efferent nerves to the oesophagus along the oesophageal nerve. It is also feasible that the horse sickness toxin fixes itself on to these nerves.

Treatment was attempted in several cases and it would seem that blistering the throat and jugular groove or intensive massage of these parts exercised no beneficial effect.

MISCELLANEOUS.

THEILER (Arnold). **Observations on an Epizootic Contagious Catarrh of the Respiratory Organs of Equines and its Relation to Purpura Haemorrhagica.**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 361–393.

In September 1916 a disease swept through the different towns of South Africa and attacked nearly all the mules and donkeys. In horses and mules it showed itself usually as a fairly benign catarrh of the respiratory organs whilst in donkeys it was very often complicated with a fulminant and frequently fatal haemorrhagic pneumonia. The epizootic appeared among the horses at the Onderstepoort Laboratory and could be traced to the introduction of a certain horse. Theiler states that a similar outbreak passed through South Africa in the beginning of 1897 at the time of the rinderpest epizootic.

The age of the horses did not appear to affect the incidence or severity of the disease and it would thus differ from strangles, for 40 per cent. of all cases were observed among aged horses. Both sexes were equally susceptible.

The period of incubation was very short, at most no longer than a week and probably as short as a day. It would thus resemble in this respect the condition described under the same name by HUTYRA and MAREK, where the period lasted from 1 to 5 days. The contagious catarrh was characterised in the main by a set of symptoms which were all present in the majority of cases, viz., fever, nasal discharge, and cough. The catarrhal changes usually originated or were localised in the upper air passages. In some cases lung changes could be clinically diagnosed or could be surmised from the course of the temperature curve. Death resulted following upon either broncho-pneumonia or purpura haemorrhagica. There was nothing typical or characteristic about the fever, it reached 104–105° F and was either simple or consisted of two successive attacks separated by a short interval. A good many of these horses were in the incubation period of horse sickness.

The catarrhal symptoms commenced with a serous discharge from both nostrils followed by fits of sneezing, early coughing with sometimes real fits of coughing. These symptoms lasted sometimes only two or three days and the horse recovered, but most frequently the disease increased in intensity and the discharge became mucous or muco-purulent and lasted three weeks or longer in many cases. Symptoms of conjunctivitis were noted in a few rare cases.

Pulmonary lesions were detected in 24 per cent. of the cases, and these revealed themselves during life by dyspnoea.

In the beginning and in light cases the horses' behaviour and appetite suffered little. Purpura haemorrhagica occurred in a few horses and the symptoms of this condition appeared very suddenly at the conclusion or during a late stage of the catarrh, that is about 24 days to a month after the onset of symptoms. Oedema of the chest, abdomen, and sheath and hind legs were seen in about 10 per cent. of all cases. Pharyngitis, lymphadenitis suppurativa, and luxation of the patella were recorded in isolated cases.

The catarrh itself did not seem to be the cause of death, but the complications following upon it, viz., broncho-pneumonia and purpura.

With regard to the differential diagnosis and identification of the disease, the question has to be considered whether it is the epizootic laryngo-tracheal catarrh of the horse described in HUTYRA and MAREK or the "influenza catarrhalis" of horses. The symptomatology of these conditions are quoted at length as given in the text books (HUTYRA and MAREK, and Wallis HOARE), and on comparison Theiler concludes that the epizootic catarrh observed in South Africa would have to be identified with that specific disease in which purpura haemorrhagica is a sequel. Thus it would neither be the epizootic catarrh nor the influenza catarrhalis described by HUTYRA and MAREK but it might be the light form of influenza of Wallis HOARE. This light influenza thus might be an entity of its own.

The experience of South Africa is stated to be of almost experimental value to explain the cause of purpura haemorrhagica, which has never been observed in the country under any conditions other than those caused by the epizootic catarrh.

THEILER (Arnold). **Dunziekte in South African Horses (Enzootic Liver Cirrhosis).**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 107–177. With 6 plates comprising 12 figs.

The South African disease of horses known as "dunziekte" has been described by observers (ROBERTSON, VERNEY) as a cirrhosis of the liver believed to be caused by the ingestion of plants belonging to the genus *Senecio*, particularly *S. latifolius*. Feeding experiments undertaken by Theiler with the plant produced, however, a parenchymatous hepatitis [see this *Bulletin*, 1920, Vol. 8, No. 1, p. 63], and thus the generally accepted view with regard to the causation of the disease would appear to be erroneous. [We recommend the reader to direct his attention carefully to the information contained in the next two extracts for further recent observations on this subject.—ED.]

In human pathology a number of conditions are described under the name of liver cirrhosis which consist mainly in an induration of the organ and an atrophy of the liver cells. The induration is due to the production of connective tissue which increases at the expense of the specific cells. Three kinds of the condition may be recognised, viz., (1) the common atrophic liver cirrhosis, Laennec's cirrhosis or granular atrophy; (2) hypertrophic liver cirrhosis (with icterus), Hanot's liver cirrhosis; (3) the cyanotic induration. The terms atrophy and hypertrophy are applied to the whole organ, which in 1 and 3 decreases whilst in 2 it increases in size. As far as the liver cells are concerned, the final result is the same in all, viz., atrophy. These three conditions represent etiologically different morbid entities, although it is not yet certain what are the causes of the first two. The third form is considered to be a simple sequel to some process obstructing the proper action of the heart.

In equine pathology no attempt has been made to differentiate between the various forms of cirrhosis. The description of the histology of the condition in this animal does not tally with that of either of the first two forms of the human lesion. The description given by KITT of cirrhosis in the horse is not sufficiently detailed to enable one to compare the condition with that of man. Since, however, identical mechanical causes probably bring about identical consequences it may be concluded that the histological changes in the indurated liver of the horse and man would be identical and thus it may be possible to find cyanotic cirrhosis, or pseudo-cirrhosis, in the horse.

The clinical symptoms of the disease described in horses, particularly of the so-called "Schweinsberger disease" which has been accurately recorded, do not correspond with those found in man. This discrepancy may be due to different causes, for example, to the difference that naturally exists between the two classes of patients. It is, however, more likely to be due to the differences in the underlying pathological changes. In man liver cirrhosis does not apparently lead to such a rapid and extensive destruction of the specific cells as it does in the horse. This fact alone may account for the difference in symptomatology, which, in the human subject, is related in the atrophic form to a stasis in the lymphatic and portal systems, but in the horse, however, to an early intoxication of the central nervous system.

A minute description is given of the morbid anatomy and symptomatology of dunziekte. Two stages of lesions were distinguished and between them there were transitions. One or both of these stages could be found in horses that had died with symptoms of the disease. The lesions begin with a stasis of the sub-lobular and central veins, leading to an extravasation of blood into the central and intermediary portion of the liver lobules. This is succeeded by hypertrophy of the walls of the veins and of the connective tissue in the lobules extending into the periphery. The induration so produced resembles to a certain extent the so-called cyanotic cirrhosis of man, but in affected horses no heart lesions could be found. The cause of the stasis in dunziekte must therefore rest actually in the liver veins. The primary stasis in these veins could be attributed to a paralysis of the vaso-constrictor nerves, or a stimulation of the vaso-dilators, which are derived from the vagus trunk. The fact that the stomach is usually enormously dilated lends support to the view that the vagus nerve is primarily attacked, and moreover, some affected animals develop grave respiratory troubles due to stenosis of the larynx, the muscles of which are supplied by the recurrent laryngeal nerve, a branch of the vagus. The formation of new connective tisssue between and within the lobules would correspond with an attempt at compensating for the lack of vaso-motor innervation of the hepatic veins. This newly-formed fibrous tissue leads to atrophy and disappearance of the liver cells. The lesions apparently are irreparable.

The disease, which appears amongst horses kept at pasture, is insidious in its onset and one of the first symptoms is yawning or gaping, together with an unthrifty-looking coat; then the horse changes its demeanour and if placed in a stable it will be noticed to develop a habit of persistently brushing against obstacles with resulting superficial abrasions on its body. Gradually, symptoms of uncertain gait become pronounced, the horse becomes restless and if let loose will flounder about. Eventually it has great difficulty in keeping its balance and staggers from side to side. A certain degree of unconsciousness appears as the disease develops. In many cases the conjunctiva has a slightly yellowish tinge, the appetite is irregular and capricious. No definite fever reactions were observed. The pulse at the beginning of the disease shows no changes but towards the end it increases in frequency and before death may reach 70 per minute. The respiration may be changed and towards the end there are symptoms of acute dyspnoea. Pregnant mares aborted towards the end of the disease. The horse tries to keep on its legs until the the very end but in some cases the animal may develop fits of real madness and become entirely uncontrollable. The disease invariably ends fatally.

Cases came under notice which ran an atypical course. The animals developed an acute attack resembling staggers. The acute form resembles cases of acute liver atrophy (acute staggers) although the underlying pathological processes are different. Apparently the absence of functional specific liver cells is responsible for the symptoms no matter what is the cause of their disappearance. In dunziekte, however, the liver lesions are not the only ones found, for paralysis and paresis of the stomach must also be considered as a characteristic symptom.

The symptomatology of dunziekte would correspond with that given of the "Schweinsberger disease" but unfortunately the histological changes in this disease have not been described in sufficient detail to enable one to identify the two conditions; but a distension of the stomach has been mentioned.

In New Zealand a disease of horses accompanied with liver cirrhosis has been described particularly by GILRUTH and is known as the "Winton disease." The symptoms manifested in the course of this disease show a striking resemblance to those of the South African condition. The morbid anatomy likewise shows certain resemblances, but the descriptions given of the lesions by GILRUTH refer not only to cases which originate in the field but to lesions found in horses and cattle which had been fed on *Senecio jacobea*, as the result of which feeding these animals succumbed. GILRUTH declared the disease so produced as identical with the Winton disease and so concluded that *S. jacobea* was the cause.

The geographical distribution of dunziekte shows a noteworthy peculiarity. In normal times it is only known to occur as an enzootic in different areas of Natal, Griqualand East, and in the District of Molteno, all situated on the South Eastern and Southern slopes of certain mountain ranges. The occurrence of the disease among military horses outside these districts can be explained by the fact that horses are known to succumb to the disease a long time after their removal from the dunziekte veld (2 years, VERNEY). The disease shows no predilection as regards sex or age but it does not seem to occur among stabled horses. In the affected areas horse breeding has now been practically all given up as the disease has increased to such an extent that horse farming has become impossible and cattle and sheep now replace horses in these districts.

The probability that the disease is caused by a nematode infestation was ruled out by feeding experiments, and likewise a considerable number of attempts to transmit the disease by inoculation failed, indicating that the disease was not a bacterial or protozoan infection. It then remained to ascertain whether the disease was caused by one or more toxic plants. Undoubtedly influenced by GILRUTH'S work on the Winton disease, South African writers (ROBERTSON, VERNEY) had looked for the cause amongst representatives of the genus *Senecio* which were numerous in the affected areas. The species *S. latifolius* and *burchelli* were subjected to trials on horses and cattle and a disease was produced which ended fatally. The lesions found were interpreted to be those of cirrhosis. As mentioned above, however, Theiler's own work in this direction failed to lend confirmation to these findings and *S. latifolius* as a cause of the South African dunziekte is therefore discarded. This conclusion naturally leads one to make a closer examination of GILRUTH'S originally recorded results, and indeed Theiler believes that the results of GILRUTH'S experiments so closely resemble those made in South Africa with *S. latifolius* that the animals died of true ragwort poisoning and not of a disease akin to dunziekte. GILRUTH'S experiments only proved that *S. jacobea* was toxic both for horses and cattle and that the disease so produced was one of the liver. If, on the other hand, the Winton disease is identical with ragwort poisoning it cannot be identical with the South African dunziekte. But upon examining the facts

Theiler maintains that Winton disease and *S. jacobea* poisoning are most likely also two different pathological conditions.

From a pathological and clinical point of view dunziekte resembles the Schweinsberger disease, which is encountered in several localities (Kurhessen, Bavaria, and the Rhine Valley in Baden). In all places it occurred on a definite type of soil, characterised by bogs, frequently exposed to inundations. In other parts the disease was connected with clover feeding but the real cause is apparently quite unknown. At all events no mention has been made of Senecio.

It is difficult to connect the etiology of the disease with any of the common causes accountable for liver cirrhosis in man. As stated above Theiler believes that the condition is set up by the action of some specific toxin on the liver nerves and that this must be looked for in one or some plants. In the feeding experiments with *Crotalaria dura* to horses and cattle (see p. 341) cirrhotic livers were found that resembled somewhat those of dunziekte. Clinically no symptoms analogous to those of dunziekte were, however, noted. But it is some other plant with more active toxic principles which must be made responsible.

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WILLMOT (Frederick C.) & ROBERTSON (George W.). **Senecio Disease, or Cirrhosis of the Liver, Due to Senecio Poisoning.**—*S. African Med. Record.* 1920. Sept. 25. Vol. 18. No. 18. pp. 346–348.

In this short paper attention is called to a disease of human beings in South Africa which is attributed to Senecio poisoning; on account of the resemblance between it and the conditions found in domesticated animals and attributed to the same origin the matter is of special interest. In 1918 certain cases of sickness of obscure causation in the George district of the Cape Province were brought to the notice of the Union Government Health Department. Whole families had suffered from time to time from a complaint, the chief symptoms of which were abdominal pain and vomiting, with ascites. It was suggested that the disease was of dietetic origin and samples of meal and wheat that had been made into bread and eaten for some time by affected persons were examined. It was found that the plants known as *Senecio ilicifolius* and *S. burchelli* grow as weeds in the wheat fields at George, and that when the wheat is threshed the seeds and portions of these plants frequently remain behind and are sold with the wheat. When the old-fashioned system of milling, with incomplete winnowing, is adopted these seeds are also ground with the wheat grains into meal. The investigators had in mind similar diseases in stock which were regarded as having been conclusively associated with plants of the same species.

The disease had existed in the George and Mossel Bay districts for at least ten years previously and during this period about 80 cases—many of them fatal—had occurred. At the time of the enquiry there were about 11 cases, the majority of which subsequently died. The patients invariably belonged to the poorer class of Europeans whose food was bread and who seldom had any other food except perhaps sweet potato.

The commencing symptoms were generally those of digestive derangement and resembled those of ordinary dyspepsia with pain in the stomach which sometimes became worse after meals. The onset was

either gradual or rapid. It would seem that the period between the onset of symptoms and death might vary from 14 days to two years or more. One man after eating for two or three days bread containing Senecio was seized with violent attacks of abdominal pain, which passed off without treatment. The symptoms continued at intervals and became more frequent and severe as the disease progressed. There might or might not be diarrhoea. Blood was often vomited or passed in the stools. The liver soon became enlarged and ascites rapidly developed in all severe cases. The temperature was generally normal or sub-normal. Signs of collapse often came on quite early in the course of the disease. There was considerable distension of the abdomen.

Most of the cases investigated were in Europeans, and the majority in children.

The liver in what seemed to be a recent case of the disease was increased in size, its edges were rounded and on its surface well-defined slightly raised areas of a deeper colour than normal were noted. On cutting into these portions they were found to be engorged with blood and to vary in size from that of a hazel nut to that of a walnut. Microscopic examination of sections showed the capillaries between the hepatic cells to be distended with blood, the central venule dilated, the liver cells reduced in size and some of them contained pigment of a brown colour and others fatty particles.

The more advanced cases showed similar sized areas in the liver but these were of a lighter colour than the liver substance and on microscopic examination the usual round-celled infiltration and formation of new fibrous tissue such as is met with in cirrhosis of the liver in other cases was the most marked change. The stomach was, as a rule, normal in size. On the mucous membrane there were numerous minute dark coloured circular spots varying in size from that of a pin's head to that of a pea. The affected area was always the larger curvature, beginning 1½ inches from the pylorus and extending along the lower part of the larger curvature for 3 inches. After washing, these dark-coloured spots appeared as very small ulcers, some of which were very superficial but others extended through the mucous coat. The bases appeared covered with haemorrhage from small eroded vessels.

With the exception of the kidneys, which showed marked congestion, the other organs examined were normal.

A number of feeding experiments were conducted upon guinea-pigs and white mice with material suspected to be contaminated with Senecio heads and also with the dried flower-heads and seeds of both *S. ilicifolius* and *burchelli* added to their ordinary food. All these animals became much emaciated in spite of the fact that they consumed a normal amount of food. One guinea-pig out of 12 died after feeding for 10 weeks on varying quantities of dried, ground-up seed heads and tops of a plant identified as *S. ilicifolius*. The post-mortem findings in this guinea-pig were almost identical with those observed in the human subject, viz., liver mottled, showing well-defined macroscopic areas of a lighter colour than normal, which on microscopic examination were found to be due to a round-celled infiltration—both intra-and inter-lobular—with the formation of new fibrous tissue. The stomach and upper part of the duodenum contained dark brown fluid and many small specks of blood were found adherent to the stomach wall, chiefly

in the neighbourhood of the pyloric end; on washing the blood away numerous minute ulcers could be made out with a hand lens.

Almost identical appearances were found upon post-mortem examination of three white rats, which died after having been fed daily for nearly four months on three grammes of ground-up heads of *S. ilicifolius*, and in one rat which, for three weeks, had had three grammes daily of *S. burchelli*.

In the three rats fed with *S. ilicifolius* the livers were contracted and distinctly granular on the surface; in the rat fed on *S. burchelli* the liver was congested but showed no evidence of contraction.

The stomach and small intestine of all the rats contained dark brown blood-stained material. Fresh blood flakes were noticed on the stomach walls in the pyloric region, and, after washing these away, very minute pin-point ulcers could be made out.

CUSHNY (Arthur R.) & WATT (Henry E.). **Senecio Poisoning.**—*Lancet.* 1920. Nov. 27. pp. 1089–1090.

"The very interesting paper by Drs. F. C. WILLMOTT and G. W. ROBERTSON in the *Lancet* of Oct. 23rd, on cases of Senecio Poisoning in man in South Africa has suggested to us that the results obtained by us some years ago may interest a wider circle of readers than those of the technical journals in which our papers* were published. At the same time some corrections and additions may be made in the accounts of our pharmacological work.

"Senecio poisoning has been known for some years to be the cause of a characteristic form of liver cirrhosis in cattle and horses, in Canada, where it is known as Pictou disease, in South Africa (Molteno disease) and in New Zealand (Winton disease). The connexion between the disease and the plants was first established by the careful experimental work of GILRUTH, CHASE, and PETHICK, an account of whose work may be found in the paper of the senior author already cited. The chemistry of the *Senecio latifolius* was investigated by one of the present writers under Professor Wyndham R. DUNSTAN's direction, and resulted in the isolation of two alkaloids, senecifoline and senecifolidine, which were examined by pharmacological experiment and were found to induce the same effects in animals as had previously been described by the veterinary investigators of the disease in cattle. Very large doses caused acute poisoning marked by excitement and convulsions from stimulation of the higher divisions of the central nervous system. But these were not of so much interest as the symptoms which followed from a single medium dose, or better from small doses repeatedly given by the mouth or subcutaneously. These corresponded closely with the disease arising from feeding the plant to laboratory animals or cattle, and also with the symptoms described by Drs. WILLMOTT and ROBERTSON. The onset was slow, no effects following the injection of a single dose for several days; the animal then began to lose weight, diarrhoea and vomiting, general weakness, staggering gait and apathy followed, developing into stupor with low temperature and final failure of the respiration. Blood was often vomited or passed in the stools and was almost invariably found in the stomach and bowel post-mortem. A certain amount of ascites was generally present in cats and sometimes this was very marked, the abdominal cavity containing large quantities of bile-stained fluid. Congestion of the stomach and duodenum with masses of black blood was present, whether the poison was given by the mouth or hypodermically. The liver was swollen and congested, and microscopically many of the liver cells were found in a state of degeneration, while the interstitial tissue was swollen and infiltrated with round cells which

* H. E. WATT: *Trans. Chem. Soc.*, 1909, Vol. 95, p. 466; A. R. CUSHNY: *Jl. Pharm. and Exp. Therap.*, 1911, Vol. 2, p. 531, and Abstact in *Proc. Roy. Soc.*, 1911, Vol. 84, p. 188.

extended between the liver cells. The whole organ presented the appearance of early cirrhosis; the small laboratory animals did not live long enough to develop the later stages described in cattle, but there could be no doubt that the symptoms were of the same character. The gall-bladder and ducts were filled with dark very viscous bile which could be pressed into the gut only with some difficulty.

"These experiments were all performed with the alkaloids of *Senecio latifolius* of South Africa, and showed conclusively that these alkaloids are responsible for the disease there known as Molteno disease. We then took up the study of the *Senecio Jacoboea* or ragwort, using samples grown in this country and in Canada, where it is the cause of Pictou disease. To our surprise these caused no symptoms in animals, and in a previous communication it was suggested that this might arise from errors in the collection or drying of the plants. Many subsequent experiments with *Senecio Jacoboea* grown in Canada or in different parts of this country from Morayshire to Surrey have always shown it to be poisonous to animals; plants collected from the stage of seedlings to the final withering and dried and prepared in different ways have proved poisonous in doses which were practically equal. And one of us has succeeded in isolating from the Canadian *Senecio Jacoboea* alkaloids, which, while differing in some chemical properties from the alkaloids of *Senecio latifolius*, resemble them in toxicity and in the symptoms induced experimentally. We are, therefore, quite unable to suggest why the first experiment failed to poison animals, but we withdraw the statements on the subject made in the previous paper. The *Senecio Jacoboea* of Canada and of this country is equally poisonous with the *Senecio latifolius* of South Africa, induces the same symptoms, and owes its toxicity to related alkaloids. Other species of the genus *Senecio* were examined. *Senecio silvaticus* proved inactive in one set of experiments, but later was found poisonous, though perhaps less so than the *Senecio Jacoboea*. *Senecio vulgaris*, the common groundsel, resembled the *Senecio Jacoboea* in toxicity and symptoms.

"Ragwort (*Senecio Jacoboea*) and groundsel are very common plants all over England and Scotland, the bright yellow ragwort giving a blaze of colour to waste lands everywhere. It is therefore remarkable that ten years ago when I made inquiries of the Board of Agriculture no case of cattle poisoning from ragwort in this country was known, though Sir Stuart Stockman was familiar with its effects in the Colonies, and had made special inquiries as to the occurrence of the disease here. During the war, however, he wrote that an outbreak had occurred in England, and one of us on examining an extract of the ragwort in the hay on which the cattle had been fed found it produced the typical action in rats. The rarity of the disease in England appears to be due to the abundant haycrop. When this is deficient, and contains an undue amount of ragwort, as occurred during the war on the farm in question, there results the same disease as is known at Pictou, where the plant is apparently widespread, and in South Africa in drought when the animals are driven to eat the plant which they generally avoid.

"The British Empire, however, does not seem to have a monopoly of this disease, for F. V. Holmboe wrote one of us in 1915 from Lyngdal in Norway, where the *Senecio Jacoboea* is very abundant, and where cattle died of a disease called "Sirasyke," which presented the characteristic symptoms of senecio poisoning. Details are given in his article in the *Norske Veterinaertidsskrift*, 1915; apparently the farmers of his district always suspected a connexion between the ragwort and the disease, and this now seems to be fairly established. Holmboe suggests that the disease also occurs in Denmark.

"The extension of the incidence of senecio disease from cattle and horses to man by Drs. Willmott and Robertson's valuable observations may draw attention to a form of poisoning which has not yet received the attention it deserves. In conclusion, it may be added that there is not the least probability of cases being met with in man in countries in which agriculture and milling are even moderately developed. Senecio is not met with in our wheat fields nor in those of the countries from which we draw our wheat, and our milling processes would remove its seeds from the corn if it were present. Senecio poison can only reach the flour when the winnowing is carried out by the most primitive methods."

THEILER (Arnold). **Geeldikkop in Sheep** (*Tribulosis ovium.*)—*Union of South Africa, Dept. of Agriculture, 7th and 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 1–55. With 7 plates comprising 15 figs.

In 1886 HUTCHEON recorded the occurrence of a disease among sheep throughout Cape Colony known to farmers as Dikgeel or Geeldikkop. The number of animals usually attacked was not considerable but the mortality among those affected was high; the disease was characterised by liver derangement, which induced a deeply jaundiced condition of the whole body, and a serous fluid was exuded subcutaneously, especially about the head region. A number of experienced farmers considered the disease to be caused by the eating of the plant "Dubbeltjedoorm" (*Tribulus terrestris*), for postmortem large quantities of the plant were found in the stomach. In 1887 DIXON, however, carried out feeding experiments with the plant, which were all negative, but from investigations in the field he concluded that the eating of the plant under certain conditions was in reality the cause, for the disease appeared only when stock were grazed in places where Dubbeltjedoorm grew in abundance. In 1906 the disease was further investigated by PAINE, who again failed to transmit the disease by feeding but obtained slight febrile reactions in attempts to transmit by inoculation; it was suggested that the disease might be malarial in nature, a view already put forward by HUTCHEON. ELLEY in 1905 likewise could furnish no evidence that the disease was due to eating the above-named plant.

From 1915 to 1917 natural outbreaks of the disease were investigated by Theiler and his staff; the disease appeared in the South-west portion of the Orange Free State, the Northern and Western parts of Cape Colony and, particularly in 1917, throughout nearly the whole of the Karroo, where it affected hundreds of thousands of sheep and caused heavy mortality.

The disease affected Merino sheep, particularly lambs, and Angora goats, together with cross-bred sheep. It did not occur every year, and it usually began in November and lasted until April. It was said to be more prevalent on River farms whilst on Berg farms few or no cases occurred; it was contracted only by sheep grazing on the veld. The low-lying parts of a farm were particularly dangerous. The earlier the first rains occurred, followed by a spell of dry weather, the earlier the disease made its appearance. If, on the other hand, no dry weather followed, few or no cases appeared.

Geeldikkop occurred earlier in the year than did blue tongue and horse-sickness, but it was more prevalent during seasons in which these two diseases were very common. However, in some years it was noticed that the disease occurred quite independently of the simultaneous occurrence of blue tongue.

Some farmers believed that the disease was caused by feeding sheep with the plant mentioned above, which sprang up after rains; healthy sheep placed on ground where this plant grew showed symptoms about two days later. Farmers held that the plants were more poisonous on lime veld than elsewhere. It was also noticed that the disease reappeared in a flock shortly after its return to the "infected" veld. On a small farm which was well stocked and closely grazed the disease

did not occur whilst on an adjoining one it was very prevalent. On one occasion it was stated that a swarm of locusts settled on the veld and ate off all the dubbeltje and subsequently no more cases of the disease were noted. Some farmers maintained that the plant was poisonous only when it became partly dry after a spell of hot weather; the seeds were stated to be dangerous only when green, but they became harmless when ripe.

Other farmers held that the insect larva found in the stalk of the plant was responsible, while others maintained that insects acted as carriers of the infection inasmuch as it was more prevalent on farms situated near the river or on low-lying parts of a farm. Others attributed the condition to sheep drinking water while in a heated condition during the heat of the midday sun.

Removal to parts of the farm situated at a higher altitude was undertaken as a preventative measure as soon as the disease appeared, and by means of this measure it was soon arrested. The disease was also noted to cease suddenly in flocks that continued to feed on the veld where they contracted it. Cases were said to develop in 48–72 hours, and no further cases were believed to occur 48 hours after the removal of a flock to clean ground. Frosts stopped the disease.

Sheep which had recovered from an attack were found liable to contract it again either during the same season or the following year. Such cases usually died. The mortality varied on different farms and in different outbreaks, and amounted to from 25 to 90 per cent.

A considerable number of experiments were made in order to establish definitely the mode of transmission of the disease. The intravenous injection of blood, pericardial fluid, and of emulsions of kidney, spleen, liver, and lymphatic gland tissue, from affected animals, to healthy sheep did not convey the affection. The drenching of sheep with portions of the intestines and stomach and their contents and of spleen and liver emulsions likewise gave negative results. Mediate and immediate contact of a sheep suffering from the disease with two healthy ones did not transmit infection.

Tribulus terrestris was obtained from three different farms; on two of these the disease was present when the material was collected while on the third no cases had been observed. The plants were dried on arrival from the first two farms but the plants used from the last farm were green. The results were all negative.

Experiments were then performed on a farm where the disease had made its appearance for three years and 700 out of a flock of 2,000 sheep had succumbed to it. On this farm the vegetation consisted mainly of composite shrub-like plants and after heavy rains the annual grasses sometimes suddenly sprang up on the plateau and gave the veld the appearance of grass land. Whilst the feeding experiments were undertaken the weather conditions for the development of the disease according to the experience of farmers was ideal. Previous to the feeding there had been an exceptional rainfall lasting for three days soaking the country thoroughly. This was succeeded by hot sunny weather which was only once interrupted, viz., by a rain on the 7th day of the experiment. *Tribulus* suddenly sprangup luxuriantly after the rain and what previously had been a dry Karroo veld changed into a luxuriant pasture; the flowering plants gave the green verdure a yellow tinge. At the same time grasses and bushes grew rapidly and well and

food became abundant. Sheep allowed to graze over this vegetation did well and returned with full stomachs every evening, while lambs kept in an enclosure and fed on *Tribulus* alone apparently relished the plant and for a time actually improved upon it. The first cases of illness that developed into Geeldikkop were noted on the 10th day, and fresh cases of the disease continued to appear up to the 16th day. The number of animals that became affected amounted to 60 per cent., and the total mortality to 45 per cent. 40 per cent. did not become affected in spite of feeding exclusively on the plant for more than a month. However, after the initial improvement all sheep lost condition.

It would thus appear that *Tribulus terrestris* is actually the cause of Geeldikkop when it is fed exclusively to sheep whilst it is green and flowering and at a time when the weather is hot.

All feeding experiments undertaken subsequently gave negative results although they were carried out under otherwise identical conditions. Perhaps some change had taken place in the plant owing to its having reached meanwhile the mature stage or, on the other hand, influences, of which a fungus infection is a notable one, were at play. The change might also be due to the weather, which was no longer so hot as during the first feeding experiments.

Theiler describes in detail the morbid anatomy, symptoms, pathogenesis, and treatment of the disease.

The body of a dead animal is usually emaciated, the visible mucous membranes are yellow, sometimes there is keratitis. The skin of the head shows changes which may be classed into two types, exudative and necrotic. In the former the serous effusion affected the skin as a whole and the sub-cutis, in the latter only the epidermis and the superficial parts of the cutis. The effusion was seen at the beginning of the disease and tended to disappear completely by the time of death. The rumen always contained ingesta consisting largely of Tribulus, the mucous membrane of the small intestines was occasionally injected, the large intestines were distended with hard balls of faeces, the liver was constantly yellow or green, the gall bladder was nearly always distended with green bile. On microscopic examination a yellow or brownish pigment was found in the liver cells, and cells that had undergone necrosis were irregularly distributed in the lobules. Tyrosin crystals were present in some of the cells. Newly-formed bile ducts were constantly present in the periphery of the lobules.

With regard to symptoms the disease was characterised by a febrile reaction during its course. This was sometimes the first symptom; sometimes it appeared only after the onset of swellings in the head. (In the climate of the Karroo, characterised by hot days and cool nights the normal morning and evening remissions in temperature in healthy sheep appeared to be more marked than in Europe,—100 or 101° to 104·5 or 105°F.). In affected cases these remissions ran up to 103 and 106°F. respectively. The fever lasted on an average about a week or ten days. The clinical symptoms were usually severe and, to commence with, were characterised either by depression or excitability. Local swellings then appeared on the head frequently in succession in different parts of the head and sometimes simultaneously. A bag-like swelling appeared under the chin and in the mandibular space ranging in size from a fowl's egg to a cricket ball. The ears were thick and pendulous. Many animals were noticed to scratch

the head. The swellings lasted from three to five days and then began to disappear. The skin then became hard and dry.

Then in about a week necrosis of the superficial layer of the skin took place with the production of extensive sores. Recovered sheep could be recognised by the mutilated ears. The second important sign was jaundice which appeared during the latter portion of the disease—commencing on from the 4th to the 10th day after the onset of the first symptoms. Whilst the jaundice increased in severity the temperature descended, the animals generally refused to eat anything, and wasting became very marked. In some instances the feet became infected, as manifested by lameness.

With regard to curative treatment experience has proved the efficacy of a purgative medicine. Apparently it does not matter what purgative is applied—calomel, jalap, or Epsom or Glauber salts. Experience also shows that a change of pasture to a place where the dubbeltjes are absent or not much in evidence immediately results in checking further outbreaks. It yet remains to be seen whether the influence of the sun is a factor required for the onset of the disease. If such be the case feeding on Tribulus might become possible if the sheep were kept during the heat of the day in a shady place and only allowed to graze during the night or early morning or late evening.

THEILER (Arnold). **Jagziekte in Horses (Crotalariosis equorum).**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. April. pp. 59–103. With 5 plates comprising 11 figs.

Jagziekte is a disease observed in horses and mules in certain districts of Natal. The symptoms exhibited are those of acute respiratory distress and the lesions consist in destruction of the epithelium of the respiratory tract resulting in emphysema and the production of granulation tissue and regeneration of bronchioles. The disease was at first considered to be infectious, chiefly on account of the lung lesions and of a definite fever exhibited during its course.

Experiments were therefore undertaken in order to ascertain whether the disease could be transmitted from sick to healthy horses by stabling them together. This led to wholly negative results. Further the injection of blood and emulsified tissue of sick animals into healthy ones failed to transmit the disease.

In November 1915 a supply of hay was received from a farmer at Allerton with the request that it be put to a test, as the disease had been noticed in the neighbourhood among horses that had been fed on the same hay. A horse was thus fed with this hay for 16 days and 279 lbs. were consumed, that is, an average quantity of 17 lbs. per day. Death occurred 93 days after the feeding has been discontinued. There was no fever during the period of feeding but a rise of temperature was noticed from the 67th to the 80th day without any concomitant symptoms.

The land from which the hay had been taken had subsequently been left to be grazed over by cattle. On inspection the grass was

found to be eaten down but the cattle had left certain bushes untouched. These unpalatable plants were identified as *Crotalaria dura*. A second feeding test was therefore carried out at the Branch Laboratory with hay from the farm containing this plant and also with specimens of the plant obtained from the farm. Positive results were obtained in four horses; although one developed only a febrile disturbance and did not develop the usual symptoms; the remaining three died.

A more exhaustive series of tests were then carried out at the Onderstepoort Laboratory in order to obtain confirmation of the above results and in order to ascertain whether the plant after heating in an autoclave would still produce the disease. From these tests conclusive information was obtained that *Crotalaria dura* is toxic for horses and is the actual cause of jagziekte. The disease could be produced both by feeding the plant contained in the hay or by mixing the plant with other food. A certain quantity had to be consumed in order to produce the disease but not all horses appeared to be equally susceptible to its toxic effects. The minimum quantity required was 46 lbs., consumed in 23 days. When larger quantities were fed for shorter periods the d ease likewise made its appearance.

Manifest symptoms were observed after a period of incubation somewhat comparable to that which is observed to take place in infectious diseases. The period varied in the different horses and its minimum length was estimated roughly at anything from 16 to 80 days. If one were to take into consideration only those experiments which gave positive results after the shortest feeding periods, the time might be fixed at about 50 days.

The disease lasted from six to 29 days. Rises in temperature were noticed in some horses before the onset of the characteristic symptoms, and these fevers were also noted in horses that did not develop the disease. In all except two horses fed on the hay and one horse that received smaller quantities of the plant a high fever was noted during the feeding period; the rise in temperature occurred immediately when large quantities of the plant were fed daily, and in about eight or ten days when smaller quantities were fed, but the fever lasted always about the same length of time.

Autoclaved plants were capable of producing the disease just as definitely as plants that had not been sterilised.

One of the most marked and characteristic lesions of the disease was emphysema. This was present in all parts of the lung both as an alveolar and an interstitial emphysema. The cut surface of the lung had the aspect of lacework perforated with holes, and the condition was particularly pronounced in the apices, where the vesicles attained from three to five cm. in di meter. Emphysema was nearly always present in the bronchial and mediastinal glands, which were enlarged and spongy in consistence, and the condition also involved the tissues surrounding the chest cavity and extended all the way up the neck and into the subcutaneous tissue of the shoulder, thoracic wall, and along the back and loins. Microscopic examination showed that all parts of the lung were involved, viz., the bronchi, alveoli, and interlobular tissue. There was very marked desquamation of the alveolar epithelium and the spaces were sometimes filled with masses of cells and liquid exudate undergoing necrotic changes. In places the alveolar

walls were completely denuded of epithelium and in other parts a considerable amount of new connective tissue was formed in the alveolar septa. This new tissue was sometimes seen to proliferate and extend into the alveolar spaces and lumens of the bronchi. A very striking lesion was the presence of proliferating bronchioles, which were grouped around a larger bronchus and were of a smaller calibre. There were no other pronounced lesions in the body. There was sometimes an increase in the amount of pericardial fluid and some liver changes comprising reduction in size, pigmentation in the central veins, slight fatty degeneration, occasionally stasis, and often the presence of newly-formed bile ducts in the periphery of the lobules.

In practice only those symptoms are usually observed from which the disease derives its name (hunted- or driven-sickness). These are noticed during the stage preceding death. In the laboratory-fed animals however, slight fevers were exhibited during the period while the horses were being fed with the plant. The fever probably formed a symptom of the disease and it might be looked upon as a series of intermittent attacks interrupted by partial recoveries, but finally ending fatally. In most cases the final fever, viz., that which was accompanied by the alarming respiratory symptoms, was the most severe one. The characteristic symptom was represented by the sudden increase in the rate of respiration which from the very onset sometimes reached a maximum of from 100 to 120 per minute and remained at this rate or only oscillated slightly around it. More frequently, however, the rate of respiration started at about 50 or so per minute and ascended gradually to 80 or more. In some cases a reverse order was noted. Sometimes the accelerated rate was alternated with periods of reduced rate. At first the movements of both thorax and abdomen were very superficial and in some horses no changes were subsequently noted. In others, however, an abdominal type of respiration gradually developed especially in those cases where the rate of respiration subsequently decreased.

Notwithstanding the extraordinary increase in the activity of the lungs the secondary symptoms were not so pronounced as, for example, those seen in an attack of horse sickness. Most of the animals had a cough and this symptom was exhibited very early. Auscultation revealed a harsh vesicular murmur which was heard in the expiratory movement, and, later, bronchial sounds, rales, and friction sounds were heard, together with symptoms indicating impaired cardiac activity. The horse's appetite was usually affected and in advanced stages the animal often showed a "tucked-up" abdomen and loss of condition. The affected animal was at first restless, but later became dull and listless. Animals usually kept on their feet until death. Farmers believe that recovery never takes place once the disease has manifested itself. From the feeding experiments it would appear that death is a certain issue when sufficient plants have been eaten.

As far as the pathogenesis of the condition is concerned one is here confronted with the remarkable fact that the consumption of a plant produced a typical disease characterised by the presence of a fever, after an incubation period. This is analogous with what is known to take place in diseases caused by micro-organisms, but these must be excluded as sterilisation of the plant did not destroy the pathogenic principle.

MITCHELL (D. T.). **The Effects Produced on Cattle by Feeding on *Paspalum dilatatum* Infected with a Species of Ergot** *Claviceps paspali.—Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 441–449.

The cultivation of the above grass on an extensive scale as a cattle feed has only recently been attempted in Natal. During the first few years the results appeared to justify the labour involved, but of late it has been noted that cattle which were allowed access to the pastures at certain times of the year develop symptoms of inco-ordination of movement. The number of animals affected is proportionately high, but the disease disappears slowly again on moving the cattle back once more on to natural veld.

Serious outbreaks had been reported from various parts of the country and four of these were investigated. Mitchell refers to the work of BROWN and RANCK of the Agricultural College of Mississippi, who have published a very exhaustive historical account of the condition known as ergotism. Six feeding experiments were performed on cattle by Mitchell himself at the Pietermaritzburg Laboratory, using Paspalum heads infected with the ripe sclerotia. As a result systematic disturbances were produced, manifested by hyperaesthesia and inco-ordination of movements, symptoms identical with those noticed in animals feeding naturally on the infected lands. The symptoms appeared rapidly after a meal and recovery was very slow. Abortion did not take place in the experimental animals, thus supporting the author's contention that this condition is not brought about in naturally contracted cases.

One meal containing 9 lbs. of heads showing a fairly high degree of infection was found sufficient to produce well-defined symptoms. The minimum quantity of sclerotia required to produce clinically recognisable symptoms was about 8 ozs. In one case the inoculation of an extract of the sclerotia produced only a transient thermal reaction. Recovery usually took place and it is believed that if animals were removed from infected lands on the first appearance of symptoms recovery would take place even without medicinal treatment. Contrary to what is the general rule in cases of poisoning by *Claviceps purpurea* ulceration and necrosis does not occur in that due to *C. paspali.*

No cases of true ergotism, due to the ingestion of grass or grain infected with *C. purpurea*, have hitherto been reported in Natal. On the other hand *C. paspali* infection is very widespread and may be looked upon as general throughout the midland portion of the Province. Outbreaks have only been reported from this portion. The climatic conditions have necessitated the cultivation of the crop on a large scale in this part, and now it is almost impossible to find an area under this grass which is not grossly infected.

Warm humid conditions favour development and dissemination of this fungus as they do other fungi, and thus the percentage of infected Paspalum heads in a pasture is very much greater in those years when a mild winter is followed by frequent rains during the spring and early summer. The stage of growth at which the fungus sets up symptoms is limited to the period when the sclerotia are ripe, and this coincides

with the ripening of the grass seeds. During the "honeydew" period no ill-effect is produced. Cattle seem to develop a fondness for the infested heads. Symptoms usually develop in about a week after exposure to the infected pasture and the percentage of animals that showed clinical symptoms varied from 5 to 60 per cent. The condition was only noted to occur in cattle, and young stock of from 14 months to 2 years appeared to be more susceptible than adult cattle. Horses, donkeys, sheep, and goats, did not develop any symptoms.

Most of the natural cases were comparatively mild:—staring coat, high-stepping movement of legs, defective vision, occasional stumbling, lateral swaying of hind quarters, and general loss of muscular tone. The appetite was unimpaired in the early stages, but later there was loss of appetite. The most acute cases were seen amongst some 18 months oxen which had been feeding on infected grass for some weeks:—partial paralysis, marked symptoms of hyperaesthesia, hurried respirations, and a rapid pulse.

The mortality appeared to be very low and no deaths were observed in the outbreaks investigated by the author himself. The loss of condition resulting from an attack is the most serious economic consideration as it requires some time for an animal to become re-established in condition after an acute attack.

Removal from infected lands, administration of a saline purgative, and feeding on green lucerne or barley are recommended as treatment. The grass should be reaped and collected during the flowering season, at which time it may be fed to cattle with perfect safety. Burning of unreaped lands is not recommended on account of the harmful effect of the fire on the grass-roots. Heavy stocking at the commencement of the year before the flowering season is advised.

A number of deaths from redwater were observed in one outbreak after nearly complete recovery from poisoning. The absence of abortion in pregnant animals and of necrosis or sloughing was noted. The condition did not give rise to any immunity or distaste for the fungus.

MITCHELL (D. T.). **A Condition produced in Cattle Feeding on Maize Infected with** *Diplodia zea.—Union of South Africa, Dept. of Agriculture. 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 427–437.

During recent years a number of cases of paralysis have been reported among cattle in certain parts of the high veld land of the country, where the animals have been allowed to graze over maize fields during the late winter months. The losses were most severe during the 1917–1918 season in the Estcourt Division and material for experimental purposes was obtained from this particular District. A few outbreaks were also reported from the Ixopo Division, but material from this District failed to produce the disease in animals at the Laboratory. When maize is harvested in Natal it is customary to reject all badly formed cobs and these are either left on the stalk or dropped on the ground. When cattle are placed on the lands these cobs are readily eaten, though they may have been exposed to the weather for several months. It was therefore suspected by farmers that the paralysis among cattle was due to eating a large quantity of

cobs that had become very mouldy. Some preliminary experiments were carried out at Pretoria with material from the Ixopo Division, with negative results, but it transpired subsequently that the amounts fed were in reality too small.

The mould infecting the damaged cobs and known as *Diplodia zea* has been described in detail by VAN DER BYL but feeding tests carried out by this worker failed to point to any harmful effects resulting from eating the infected cobs.

Subsequently, Mitchell obtained a quantity of the mouldy cobs from the Estcourt Division and tested their effects upon four oxen at the Pietermaritzburg Laboratory. These oxen, prior to the experiment, had been fed only on grass in paddocks adjoining the laboratory, were five years old, and in good condition. During the experiment they were tied up in the stable and watered by hand with water obtained from an adjoining river. The maize was fed just as it was received from the farmers, but only the grains were eaten and the central core was always rejected. It was, therefore, difficult to say what weight of material was actually eaten.

This experiment showed that a condition indistinguishable clinically from that occurring naturally on the infected mealy lands could be set up artificially by feeding on the cobs. One animal died as a result of the feeding but it was thought probable that this animal would have recovered if the feeding had been discontinued altogether during an interval in the course of the experiment when the animal was showing marked improvement. In the other three animals, as soon as partial paralysis or incoordination of movement was observed no more mouldy cobs were fed. The quantity of cobs fed was considerable. In the case of the ox that died it amounted to 225 lbs., but the other animals were fed with even larger quantities. The general health of the animals did not seem to be greatly affected; the appetite was maintained; there was slight diarrhoea in the earlier stages, and constipation in the later stages, and no fever.

An attempt was then made to ascertain whether the trouble was caused by the mould itself or by some product formed due to its multiplication in the corn cob.

Sterile maize kept in sterile jars was inoculated with Diplodia and incubated at 27° C. for 2 months when massive quantities of spores had become formed. Twenty pounds of this infested material were given to each of three cattle and two of them developed symptoms. These sometimes resembled those observed in the preceding experiment, but their onset after the commencement of feeding was more rapid and they were more acute in character. Also the period during which symptoms were shown was shorter and recovery more rapid. These differences are accounted for by the fact that the animals received a large dose of more highly concentrated material than did the first lot of animals. The one animal which did not develop symptoms received a larger dose than either of the other two, but this animal might have acquired a tolerance from having recovered from an attack in the preceding year.

Further experiments proved that cultures of an allied mould (*Mucor mucedo*) grown in maize media were incapable of producing clinical symptoms such as the above, and that when the Diplodia was grown on a medium containing a minimum quantity of starch and sugar

(finely chopped hay covered with a little oatmeal porridge) it was harmless when fed to cattle.

In natural circumstances the disease is limited to the late winter months, July to December, when veld grazing is scanty and farmers turn their cattle into the maize lands for better feed. Cattle only appear to be affected, but there is no predilection as regards age and sex. Cases were known to occur in 6–8 days after the animals had been placed on these lands. In the Laboratory the shortest time which elapsed between the commencement of feeding and first symptoms was three days and the longest 15 days. The development of symptoms appears to depend upon the quantity eaten and the degree to which the cobs are infected.

The first symptoms were lachrymation and salivation, slight quivering of the flank and shoulder, slightly arched back and standing with the legs further apart than normal. The head was carried lower and the ears drooped slightly. The faeces were rather soft. Slight inco-ordination of movement was noticed on walking the animal.

Later, these symptoms became aggravated—profuse salivation and lachrymation, muscular tremors, staring coat, marked arching of the back, dejected appearance, difficulty in moving, high-stepping movements when made to walk, and lateral swaying. These symptoms lasted one or two days and subsequently the animal was unable to rise without assistance. Later these changes became still more marked and death was preceded by a complete loss of tone of the muscles. If the feeding was discontinued when well-marked symptoms had developed recovery was fairly rapid, the appetite returned, and the animal fed on green stuffs quite freely. Symptoms of paralysis disappeared in a few days.

In one case examined post-mortem the chief lesions found were a well-marked catarrhal enteritis affecting both the small and large intestines, acute diffuse hyperaemia of both kidneys, and well marked congestion of both lungs. The mortality varies considerably on different farms. On some farms nearly all the animals that develop symptoms succumb and on others a large percentage recover. The mortality appears to be greater during the later part of the season. No treatment other than good nursing and the administration of a saline purge is recommended, and for purposes of prevention, all cobs should be harvested from the lands.

GREEN (H. H.) & DIJKMAN (C. D.). **Some Experiments on the Fate of Arsenic in the Animal Body.**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 689–698.

In view of the great extent to which arsenic preparations are now employed for the dipping of stock in tropical and sub-tropical countries the data recorded by these authors are of considerable practical importance. Moreover, the determination of the distribution of small quantities of arsenic in tissues was rendered possible by a method of analysis described by Green [this *Bulletin*, 1920. Vol. 8. No. 1. p. 89], the so-called micro-titration method.

In general the healthy skin absorbs a small amount of salts applied to it in solution, and in the dipping of stock in arsenical baths a small

amount of arsenic find its way into the blood stream and is subsequently eliminated, chiefly in the urine. The absorption from abraded or injured surfaces is much greater.

The only previous work dealing with the elimination of arsenic in the urine after dipping or spraying appears to be that of WATKINS PITCHFORD (H.). Green, however, considers that the numbers furnished by this author are on physiological grounds inordinately high, especially a figure such as 0·097 per cent., which would seem to indicate an acute arsenical intoxication. Green computes that such figures would make the amount eliminated by the urine in 24 hours amount to well over 2 grammes, a quantity which if it passed straight into the blood stream in a soluble form might easily prove fatal.

Green and Dijkman therefore collected urine at certain intervals from two regularly dipped oxen, one of which had been dipped at three-day intervals for two months, and the other at five-day intervals for four months. The results obtained by analysing the urine would indicate that PITCHFORD's figures were from 40 times to well over 100 times as high as the authors' figures. Thus for the urine passed 10 hours after dipping his figures ranged from 0·028 to 0·097 per cent. arsenious oxide, and theirs from 0·0005 to 0·0009 per cent. It was estimated that the actual elimination of arsenic by this channel was extremely small, ranging around $\frac{1}{10}$th of a milligramme per hour.

The urine of a horse dipped at regular weekly intervals in arsenite of soda equivalent to 0·16 per cent. arsenious oxide, was also subjected to examination. The amount eliminated was again estimated to be always very small, ranging from about $\frac{1}{10}$th to $\frac{1}{3}$rd of a milligramme per 100 c.c. or only up to a maximum of 23 milligrammes in 24 hours. The amount of arsenic eliminated per day a week after dipping was less than that eliminated during the first few days, but the falling off was not so great as might be expected, either because when such small amounts reached the blood stream elimination is protracted by temporary fixation in the tissues, or because arsenite which is ostensibly dried up on the scurf and hair continues to be absorbed by the skin.

Arsenic could also be detected in the faeces of dipped cattle.

The figures given by PITCHFORD for the amount of arsenic fixed in the hides of dipped animals also appeared to the authors to be much too high, and unjustifiable conclusions are regarded to have been drawn from the data by COOPER and LAWS, 1915, in an article entitled "Some observations on the theory and practice of Dipping." Three sets of analyses were made by Green and his co-worker (1, 6, and 14 days after the last dipping of animals that had been dipped every five days for three months). The discrepancies in results obtained were again very striking. For the shaved hide, or skin proper, PITCHFORD's figures were from 40-70 times as high as the authors', i.e., from 137 to 251 milligrammes per square foot as against 4 milligrammes. The only difference between the methods of treatment was that the authors' cattle were dipped in plain aqueous arsenite of soda (0·16 per cent. arsenious oxide), whereas in PITCHFORD's dipping soap and paraffin were also present. PITCHFORD's data would make the total amount of arsenic fixed in the shaved hide, or skin proper, to be about 7-12 grammes, while the present figures worked out only to 0·16 to 0·2 gramme. The small amount of fixed arsenic recorded was much the same ten days after dipping as it was one day after dipping.

The differences recorded for the hair and scurf were deemed less worthy of comment for the arsenic concerned was mainly arsenite which had dried on and the presence of soap in PITCHFORD'S dip might account for the high figures for merely adherent arsenic. For the sake of getting an approximate idea of the amount of arsenic adhering to the hair and scurf the carcase of another regularly dipped ox was examined. One square foot on the hind quarter was marked off, clipped, shaved, and then cut out. The clipped hair was soaked in water for half an hour, filtered and washed. The filtrate showed 0·052 gramme arsenious oxide and the rinsed hair 0·028 gramme per square foot. The residue of short hair and scurf removed by shaving (with soap) showed 0·011 gramme arsenious oxide per square foot more or less "fixed" and 0·035 gramme readily removed by washing with water. The greater proportion of the arsenic in the hair and scurf (total 0·126 gramme per square foot) was therefore not fixed but merely adherent. The shaved hide, or skin proper, showed only 0·006 gramme arsenious oxide per square foot, of which 0·0033 was present in the epidermis and 0·0027 gramme in the underlying connective tissue.

Green and Dijkman next performed some experiments in order to determine the rate of elimination of arsenic after oral dosing and after intravenous injection.

To determine the rate for oral dosing two experiments were carried out, one on a sheep and one on a horse, in which known doses of sodium arsenite (equivalent to 0·2 grammes arsenious oxide for the sheep and 2 grammes for horse) were administered and the daily elimination in faeces and urine followed for eight days. For the sheep the elimination in the urine rose for the first 48 hours, and then diminished until at the end of eight days it was down to 1 milligramme for the whole 24 hours output. By this time 93 per cent. of the dose had been excreted, of which 79 per cent. appeared in the urine and 14 per cent. in the faeces. With the horse, on the other hand, the greater part of the dose was eliminated in the faeces (23 per cent. in the urine and 59 per cent. in the faeces), altogether amounting to 82 per cent. of the dose. This suggests less perfect intestinal absorption of arsenic in the horse than in the sheep. A supplementary experiment confirmed this view.

Four experiments were carried out in order to determine the rate of elimination of arsenic after intravenous administration, and three to determine and compare the distribution of ingested arsenic over the tissues. The first series, however, miscarried and have to be repeated. In the second series it was found that in the cow, sheep, and horse, the liver tissue fixed most arsenic per unit mass, but that the muscular tissues owing to their dominant bulk had fixed the largest absolute amount. The fixation by the kidneys was also high when expressed per unit mass, but that by the blood low and by the brain, nerve, and bone extremely low. Small but easily detected quantities also appeared in the faeces.

Data are next given for arsenic in the alimentary tract, liver, spleen, and kidneys of a horse destroyed by oral administration of arsenic.

Some estimations were made upon the toxic dose of arsenic for the various animals. The figures usually accepted are those of KAUFFMAN. Green and Dijkman found by experiment that one gramme arsenious oxide as sodium arsenite absorbed into the blood stream might easily

prove fatal for the horse although occasionally horses could stand appreciably higher amounts. When arsenic was administered orally the results were uncertain. In one case, however, a drench containing six grammes arsenious oxide as arsenite was given by the mouth with no apparent ill-effect. With regard to the arsenious oxide itself one horse was dosed with 1 gramme of the powder and another with 2 grammes of the powder every day for a fortnight without suffering any ill-effects. Another horse received 4 grammes daily and died by the time 24 grammes had been administered. A fourth received 5 grammes twice weekly and died after the fourth dose.

In experimental work on sheep it was found that death was uncertain with anything less than 0·5 gramme arsenious oxide as arsenite (10 milligrammes per kilo) administered orally, or 0·3 gramme (6 milligrammes per kilo) intravenously. In most cases 0·2 to 0·3 gramme was safe by the mouth (5 milligrammes per kilo), and experience in the treatment of *Haemonchus contortus* infestation in flocks showed that 0·1 gramme (0·2 to 0·3 milligramme per kilo) could be safely administered medicinally. 0·1 gramme was injected intravenously without producing symptoms of intoxication and even 0·2 gramme was injected into three sheep without disaster. 0·3 gramme injected intravenously was rapidly fatal in two cases, but non-fatal in a third, although symptoms of intoxication were shown.

As regards cattle 1 gramme (2 milligrammes per kilo) would appear to be moderately safe by intravenous injection while 2 grammes is uncertain. The effects of oral administration of soluble arsenic are more erratic with cattle.

GREEN (H. H.) & KESTELL (N. H.). **Behaviour of Bacteria towards Arsenic.**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 701–706.

"*Summary.* Differences in tolerance of different bacteria for arsenic are very marked. Many which are fairly tolerant of arsenate are relatively sensitive to arsenite. Certain groups are characteristically sensitive, *e.g.*, the *subtilus* group, of which the four leading representatives were tested and all found intolerant of 0·05 per cent. of As O as sodium arsenite in broth. Other groups, notably the *putidum* family, can tolerate from ten to twenty times this concentration, some members growing freely in 1 per cent. arsenite broth. The *colon-typhoid* group is sensitive as a family but has at least one outstanding exception in *B. arsenreducens*, and other resistant members probably exist. Resistance to arsenic is, therefore, not a rigorous group characteristic, although it is probably as characteristic as any other biochemical feature and might find a place in diagnostic bacteriology. Apart from the members of the putidum group five highly resistant *bacteria* (*i.e.*, non-sporulating rod forms) are described, but not named.

"Of four *cocci* tested three were found sensitive and one tolerant. Two members of the *streptothrix* group were both sensitive. *Yeasts* and *moulds* generally show a high degree of tolerance.

"Although over a dozen arsenic resistant species of bacteria were examined, only two showed any chemical activity towards arsenic; the earlier described *B. arsenoxydans* which oxidises arsenite to arsenate and *B. arsenreducens* which reduces arsenate to arsenite. The others are merely tolerant. There is no discernible relationship between arsenate reduction and nitrate reduction.

"Arsenite resistant bacteria are infrequent in soil but fairly frequent in faeces. About 10 per cent. of the bacterial count of fresh stable manure were found moderately tolerant, and about 1 per cent. highly tolerant. In arsenical dipping tanks an automatic enriching of resistant faecal bacteria, and suppression (or metamorphosis ?) of sensitive forms takes place."

DE KOCK (Gilles van de Wall). **A Preliminary Note on the Toxic Effect of Methylene Blue.**—*Union of South Africa, Dept. of Agriculture, 7th & 8th Repts. of the Director of Veterinary Research.* 1918. Apr. pp. 677–685. With 3 charts.

The selective staining affinity of several of the aniline dyes for several of the pathogenic organisms suggested originally to PENZOLDT the idea that they might be used as antiseptics and disinfectants, and STILLING introduced several into therapeutic use. Many, including methylene blue, have been found to have to a certain degree an antiseptic action, but, with the exception of a few like brilliant green, their use has not met with any appreciable degree of success.

Methylene blue was first tried in the treatment of equine biliary fever at the Onderstepoort Laboratory in 1913 (see p. 288), for unsatisfactory results had been obtained with the more common substances used in the treatment of protozoan affections. Even trypan blue, which appears to act as a specific in canine and bovine piroplasmosis, seemed to have no effect on piroplasms of the Nuttallia type. When injected intravenously in a sufficiently large dose (500 c.c. of a 1 per cent. solution) methylene blue, however, seemed to have a specific effect on these parasites. Two cases were treated satisfactorily in 1913, and nine were subsequently injected (1 very acute, 5 acute, 3 mild); but three of these developed a febrile reaction which ended fatally, although parasites could no longer be found in their blood after the 4th day. On account of the deleterious effects resulting from the injection of massive doses it was decided to try smaller doses; but no apparent effect was thereby produced upon the parasites and one case died of biliary fever. It was at first thought that animals so treated showing subsequently secondary fever reactions had become infected with the virus of infectious anaemia and hence it was considered necessary to make further enquiries as to the action of methylene blue.

Two normal horses were injected intravenously with 500 c.c. of a 2 per cent. solution. Both commenced to react a few days after the injection, showed fairly acute symptoms of anaemia with irregularly remittent fever, and were finally killed in extremis.

Further investigations were carried out on a number of normal horses, which were fairly young and in prime condition, in order to find out to what extent the drug produced febrile reactions and symptoms of anaemia when injected intravenously in large doses, and whether the toxic substance present in the drug could be destroyed by sterilisation. Two lots comprising three horses each were injected respectively with unsterilised and sterilised solutions (500 c.c. of a 1 per cent. solution on two consecutive days). The results were the same in all the horses. They all showed symptoms of anaemia, in some resembling those seen in cases of biliary fever and in others those seen in certain forms of equine infectious anaemia. All with the exception of one developed febrile reactions. In some the fever

commenced on the day of injection while in others there was an incubation period. The fever was again of an irregularly remittent nature, preceded or followed by frequent exacerbations of temperature which sometimes lasted for several months. Symptoms of anaemia usually became apparent two or three days after treatment. The animal appeared to be dull and restless, did not feed well, and the conjunctival and buccal mucous membranes were distinctly pale, with a yellowish tinge. There was rapid loss in condition, the pulse increased in frequency, up to 112 per minute, and subsequently the affected animal began to drag its hind legs, swayed from side to side, staggered and lay down. Cases which did not succumb made a slow recovery, in about 2–3 months. Blood smears from most of the animals showed signs of anaemia in the form of anisocytosis and in one case poikilocytosis. At the commencement serum from the horses was tinged with haemoglobin but a few days afterwards it became clear again. In most horses the urine was dark blue in colour and remained of this colour for a few days. The number of red corpuscles dropped to three millions per cubic mm., and even less, in a few weeks. The haemoglobin content was also reduced.

Two of the animals died, one on the 6th day and the other on the 12th day after injection. The first of these was also suffering from purulent nephritis. On post-mortem examination lesions of acute anaemia were observed—watery condition of the blood, blanched appearance of the mucous membranes and other tissues, etc.

Blood was transfused from these animals into normal horses at different stages of the disease in order to determine whether they might have become affected with infectious anaemia accidentally. These experiments proved negative.

Again the effects of methylene blue were tested by intravenous injection of large doses into cattle and sheep. The cattle each received 500 c.c. of a 1 per cent. solution on two consecutive days and the sheep 100 c.c. of a 1 per cent. solution on two consecutive days. Similar results were observed. In one bull the reaction that followed was acute and commenced with a rise of temperature on the 7th day after injection, which reached 106°F. on the 10th day; this animal died from toxic anaemia.

De Kock concludes from these facts that methylene blue cannot be regarded as a non-poisonous inert substance which can be used in any quantity without ill-effects. The clinical picture produced by the substance resembles so closely that of equine infectious anaemia that it is impossible to differentiate the two conditions without employing the usual biological test, viz., blood inoculation.

Erratum.

1919. Vol. 7. No. 4. p. 248. 15th and 14th lines from bottom. For "an ulcerous stomatitis of cattle was met with both in calves and adults while being fed on 'safari' (a coarse herb seen at the end of the dry season)" *read* "an ulcerous stomatitis . . . fed during a journey."

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